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The Australian and New Zealand Journal of Surgery

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PERCIVAL POTT AND HIS PUFFY TUMOUR.

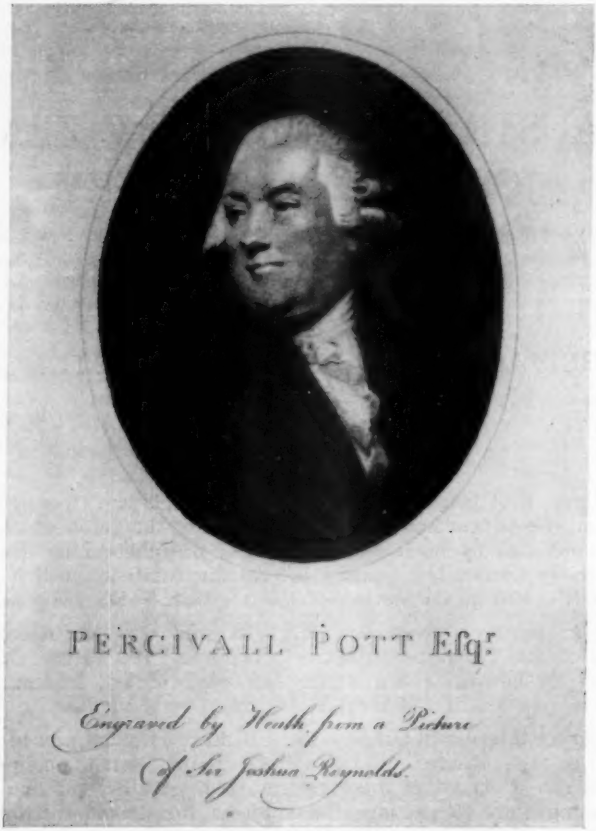
By K. F. RUSSELL,
Melbourne.

PERCIVAL POTT, that great surgical lion of the eighteenth century, needs no introduction. He is best known by the fracture of the ankle which he himself sustained, and also by his masterly account of tuberculous disease of the spine. A lesser known but equally interesting condition which he was the first to describe will be the subject of this memoir, Pott's puffy tumour.

Pott was born on December 26, 1713, and was the son of Percival Pott, Esq., of old Threadneedle Street, London. His father dying shortly after his birth, he was taken under the wing of Dr. Wilcox, Bishop of Rochester, a relative of his mother.

Showing an interest in surgery early in his youth, he was in 1729 bound apprentice to Mr. Nourse, a surgeon to Saint Bartholomew's Hospital. Mr. Nourse ran a school of anatomy in Aldersgate Street, and it was the duty of young Pott to prepare the subjects for demonstration. By this means he obtained that groundwork of anatomy so necessary to the practice of surgery. His clinical knowledge he collected in the wards of Saint Bartholomew's Hospital under Nourse's eye. At the age of twenty-two, in 1736, his apprenticeship being completed, he entered private practice, and so quickly made a name for himself that in 1744–1745 he was elected assistant surgeon to Saint Bartholomew's Hospital. His appointment as full surgeon followed in 1749. In 1756 he suffered the fracture of the ankle which now bears his name, the story of the accident being too well known to retail here. The fracture being a compound one, his foot was in danger of amputation, but the efforts of his old tutor to save the limb were successful.

During this period of enforced inactivity he planned his "Treatise on Ruptures", which was completed by the end of the year. This was followed in 1757 by an account of the "Hernia Congenita", which resulted in some dispute with William Hunter, who claimed priority for the account, but after some discussion on either side the dispute terminated. His various papers which followed are set out below, with the year of their appearance.



1758. "Observations on the Fistula Lacrymalis."
1760. "Paper on the Nature and Consequences of Wounds and Contusions of the Head, Fracture of the Skull, etc."
1762. "Practical Remarks on the Hydrocœle."
1764. "Case of Hernia of the Urinary Bladder", published in *The Philosophical Transactions of the Royal Society.*
1765. "Account of the Fistula-in-Ano."
1772. "Remarks on the Passing of the Seton."
1775. "Chirurgical Observations. A Collection of Remarks on Many Surgical Conditions."
1779. "Remarks on that Kind of Palsy of the Limbs which is Frequently Found to Accompany a Particular Curvature of the Spine." This, his greatest work, was a classic on the signs and symptoms, pathology and treatment of tuberculous disease of the spine. It was supplemented by a further account in 1783.

In 1764 he was elected a Fellow of the Royal Society. In 1786 the Royal College of Surgeons of Edinburgh honoured him by making him their first Honorary Fellow, which appointment was followed in 1787 by an Honorary Diploma from the Royal College of Surgeons of Ireland.

In this latter year he retired from the staff of Saint Bartholomew's Hospital, and on December 22, 1788, he died in his seventy-fifth year from pneumonia following a chill caught when he was visiting a patient twenty miles from London ten days previously.

The description of the puffy tumour which now bears his name is contained in his paper on "Wounds and Contusions of the Head". In describing the occurrence of extradural and subdural hæmatoma and the possibility of infection supervening, he goes on to say:

> In order to explain my meaning as clearly as I can, I will consider the inflammatory effect of contusion by itself. . . . If there be neither fissure nor fracture of the scull, nor extravasation . . . . and the scalp be neither considerably bruised, nor wounded, the mischief is seldom discovered or attended to for some few days. The first attack is generally by pain in the part which received the blow. This pain, though beginning in that point, is soon extended all over the head, and is attended with a languor, or dejection of strength and spirits, which is soon followed by a nausea, and inclination to vomit, a vertigo or giddness, a quick and hard pulse, and an incapacity of sleeping, at least quietly. . . .
>
> A day or two after this attack, if no means preventative of inflammation are used, the part stricken generally swells, and becomes puffy, and tender, but not painful; neither does the tumour rise to any considerable height, or spread to any great extent: if this tumid part of the scalp be now divided the pericranium will be found of a darkish hue, and either quite detached, or very easy separable from the scull, between which and it will be found a small quantity of a dark-coloured ichor.
>
> If the disorder has made such progress that the pericranium is quite separated and detached from the scull, the latter will even now be found to be somewhat altered in colour from a sound healthy bone.
>
> With further advance of symptoms . . . . the alteration of the colour of the bone will be found to be whiter and more dry than a healthy one, or, as Fallopius has very justly observed, it will be found to be more like a dead bone. . . .
>
> If the dura mater be denuded, it will be found to be detached from the inside of the cranium, to have lost its bright silver hue, and to be, as it were, smeared over with a kind of mucus, or with matter, but not with blood. . . .
>
> If the bone be perforated matter will be found between it and the dura mater, generally in considerable quantity, but different in different cases and circumstances. Sometimes it will be in great abundance, and diffused over a very large part of the membrane; sometimes the quantity will be less, and consequently the space which it occupies smaller. Sometimes it lies only on the exterior surface of the dura mater; and sometimes it is between it and the pia mater, or also even on the surface of the brain, or within the substance of it.

The details in this memoir and the illustration accompanying it are taken from the 1790 edition of the collected works of Percival Pott, edited by James Earle, Esq., in three volumes. The volumes are in the possession of the Medical Library, University of Melbourne.

CEREBRO-CRANIAL INJURIES.[1]

By GEORGE W. SWIFT,
Seattle, Washington, United States of America.

INTRODUCTION.

THE problem of severe cerebro-cranial injuries has developed within the last quarter of a century, concurrently with rapid transportation and the increased use of machinery in industry. Twenty-five years ago a "skull fracture" was comparatively rare; gunshot wounds of the head were about the only traumatic lesions the surgeon was called upon to treat. Today, however, he constantly meets the problems consequent on cerebro-cranial injury.

The automobile has had a definite effect upon the entire social fabric of modern life. Our population has been redistributed; cities have been remodelled and suburban communities have supplanted civic centres; broadened thoroughfares have replaced narrow tortuous streets; laws regulating everything pertaining to the automobile, street traffic and pedestrians have been devised. All these changes have led to an increase of speed in transportation and, in turn, to a greater number of accidents. What has happened in the case of the automobile has also occurred in industrial activity. Speed in the production of raw materials, such as lumber and minerals; speed in the mass production of factories; speed in the distribution of the finished product, in the loading of trucks, ships and cars—speed everywhere has resulted in countless injuries.

As the nervous system of the individual member of society has responded in varying degrees to this mania for speed, the inexorable law of the "survival of the fittest" has come to play its rôle in a complicated, daily routine life of the individual, often with tragic consequences. Somewhere in this picture is hidden the fascinating story of the development of both the cause and treatment of severe cerebro-cranial injuries. Somewhere is shown the importance of the care given not only immediately after the injury, but also that given during the ensuing days or weeks.

Records of severe head injuries in the past two decades show both the increased frequency of these injuries and the decrease in mortality (Swift[(1)]) (see Table I). From these studies it is evident that three factors enter into the problem of decreased mortality: (1) care of the patient, (2) compilation of statistics, (3) publication of results in various localities by leaders in this field.

In considering these factors that have a bearing upon cerebro-cranial injuries, the surgeon has not had to depend upon individual effort alone. The American College of Surgeons, through its Board of Traumatic Surgery and Industrial Medicine, has been able to interest large industrial concerns, insurance companies and traffic agencies in the prevention and care of these

[1] Read at the annual meeting of the Royal Australasian College of Surgeons, held at Sydney, March, 1938.

TABLE I.

Showing Increased Incidence and Lowered Mortality in Head Injury Cases during a Period of Eighteen Years.

Period.	Number of Cases.	Death in Three Hours.	Death Within Twenty-four Hours.	Death After Twenty-four Hours.	Total Mortality.
1919 to 1924—60 months ..	178	—	—	—	60·7%
March, 1931, to December, 1932—21 months	175	—	—	—	23·3%
October, 1934, to March, 1935—6 months	214	—	—	—	19·1%
October, 1936, to March, 1937—6 months	145	—	10 66·0%	5 33·0%	15 10·3%
October, 1937, to December, 1937—3 months	107	4 30·0%	10 77·0%	3 23·0%	13 12·1%

accidental cases. The American Medical Association, through its Committee on "Fractures of the Skull", has made a comprehensive study of the problem in many cities and in rural districts, and each year has presented charts and tabulations at its annual meeting. Added to the efforts of various organizations has been the work of many individual leaders. Dr. Harry Mock,[2] of Chicago, has delivered hundreds of lectures to various component societies of the American Medical Association, explaining the treatment of injuries involving the brain. Perhaps the most outstanding contribution to this great work is that on water balance in relation to the cerebral circulation, carried out by Dr. Temple Fay[3] and his associates in Philadelphia. Lastly, Dr. Walter Dandy,[4] of Baltimore, has contributed the procedure of encephalography, which is used for both diagnostic and therapeutic purposes in the prevention and cure of many post-traumatic sequelæ in patients formerly allowed to develop into a condition of hopeless invalidism. In addition to these three men, who have thus shared in the victory in the control of this problem, other earnest workers, by reporting their successes and failures, have added to the sum total of our present knowledge of this subject. Although it is the purpose of this paper to review the problem as we have seen it developed in the Pacific North-West, we have taken freely, from any and all, suggestions that appealed to us in the care of these cases, and our results should reflect the average throughout the country (Jackson,[5] Holbrook[6]).

THE IMMEDIATE TREATMENT OF CEREBRO-CRANIAL INJURIES.

The treatment of all severe cerebro-cranial injuries consists of two major divisions: (1) the immediate relief of shock and the treatment of injured brain tissue, and (2) the treatment of post-traumatic sequelæ. The immediate care is usually given by the first surgeon called, but the more prolonged, post-traumatic care should be under the direction of a surgeon qualified by

experience in such cases. The more or less arbitrary separation of the treatment into two periods may not be accepted by surgeons at large as at all practical, but the suggestion is at least worthy of serious consideration and discussion. Perhaps eventually a definite routine or standard method of handling these cases in the individual hospital will become more universal, and the responsibility will then rest upon the hospital staff. Certainly publication of reports of end-results will stimulate a better appreciation of the importance of the first period of treatment both by the staff members and by the individual surgeon.

The decision as to when the first period has ended and the second begins must be made according to the individual case. Some surgeons classify the periods or stages as those of (1) shock, (2) acute œdema, (3) chronic œdema, and (4) convalescence (Ochsner[7]). Whatever plan or division into degrees or stages may be used, it is our opinion that the cases fall naturally into two periods: the first requiring immediate attention, and the second requiring more considered and perhaps experienced care.

In the immediate care of an injured patient many factors are involved, although simple measures suffice in the less seriously injured. Since accidents may occur at any time or place, and since the character as well as the severity of the accident becomes important and may be the decisive factor, the first thing to determine in any case is whether the patient is in immediate danger of death. This is easily done. Three things should be ascertained: (1) Is there severe hæmorrhage either intracranially or extracranially? (2) Is the patient in severe shock? (3) Is the spinal cord severed? A simple examination, which can be made without any unnecessary moving of the patient, will answer these questions.

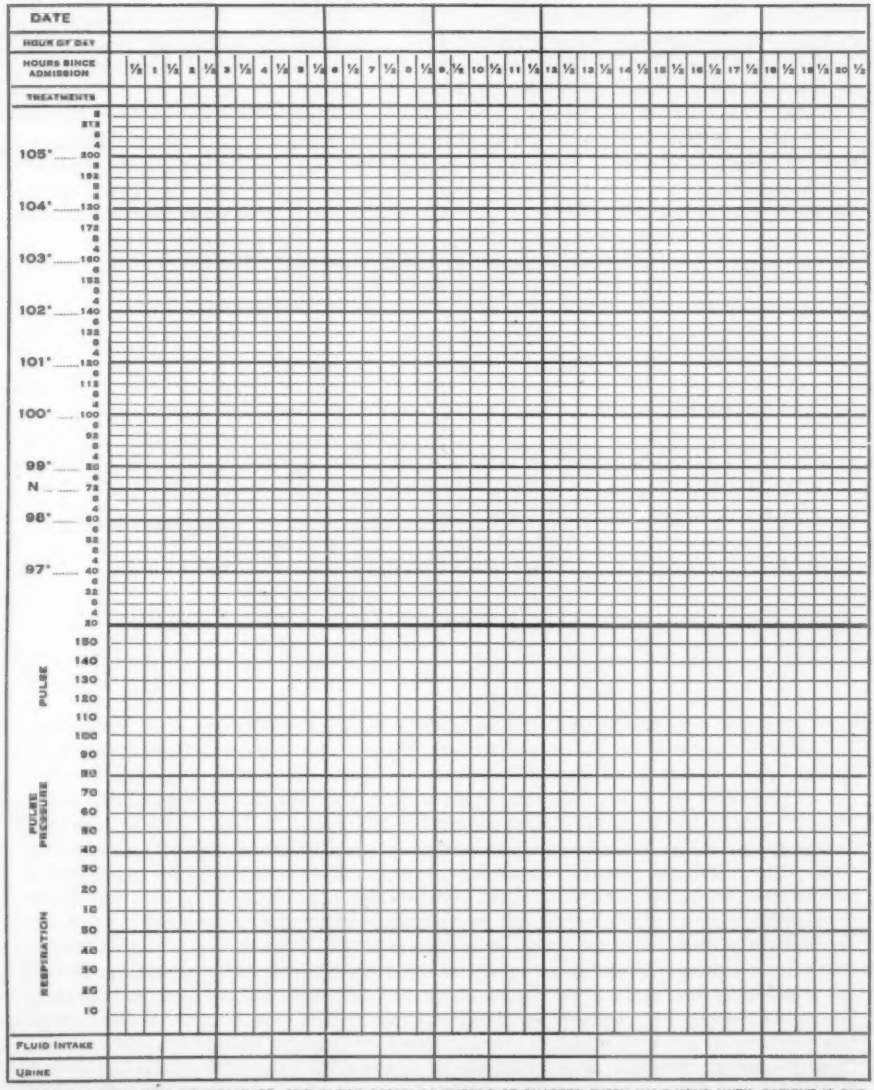
Hæmorrhage.—Hæmorrhage requires immediate action. If extracranial, its source is usually scalp vessels which can be easily compressed by sterile gauze and bandage. If from the nose or mouth it is more difficult to control. Elevation and rotation of the head so that the blood will escape from the nose or mouth are usually sufficient to control this type of bleeding. Intracranial hæmorrhage is more difficult to diagnose and also to control. A massive hæmorrhage promptly reveals itself both in the character of the pulse, which is at first rapid and weak, then progressively firmer and slower, and in the character of the respiration, which is initially slow and stertorous and later shallow and of Cheyne-Stokes type. These patients rapidly become cyanotic and death occurs within a few hours. Less severe hæmorrhages fall into several groups: (1) middle meningeal, extradural and subdural, (2) chronic subdural hæmatomata, (3) subarachnoid hæmorrhages, either basilar or cortical, (4) subcortical, and (5) ventricular. Spinal puncture should be used to diagnose and differentiate the types. Encephalography may be necessary to make a positive diagnosis. One is rarely justified in surgical interference until the localization is complete.

Shock.—What spinal puncture is to the diagnosis of hæmorrhage, blood pressure estimation is to the diagnosis of shock. So many factors complicate the picture that this accurate test is essential. When the systolic pressure is below 50 millimetres of mercury and the pulse pressure below 20, the patient is in shock. The colour of the skin is not a reliable test, since it varies greatly through many causes. Vasomotor changes may also be due to conditions other than intracranial and may be misleading. Respiration

THE NEURO SURGICAL CLINIC
SEATTLE

GRAPH—A

NAME SERVICE WARD CASE



PULSE, RESPIRATION, TEMPERATURE, AND BLOOD PRESSURE SHOULD BE CHARTED EVERY HALF HOUR UNTIL PATIENT IS OUT OF SHOCK. CHART ALSO: SPINAL PUNCTURES (I), INTRAVENOUS GLUCOSE (G), MAGNESIUM SULPHATE DEHYDRATION (D), ATROPINE (A), PITUITRIN (P) EPHEDRINE (E), CAFFIENE (C) MORPHINE (M) AND SEDATIVES (S), AS GIVEN IN "TREATMENTS"

CHART I. From the Neurosurgical Clinic, Seattle. Half-hourly chart.

and pulse regularity, too, may be unreliable. Blood pressure readings, therefore, must be relied upon to determine this question accurately. Repeated readings every half hour for the first twelve hours are necessary to keep an accurate picture of the case. In our chart we have a special partition for pulse pressure as well as for blood pressure, temperature and pulse readings (see Charts I and II). A glance at these charts at any hour reveals the true condition of the patient. My associate, Dr. Berens,[8] has written on the subject of the cause of shock and the problem of water balance in the control of shock as we understand it.

Treatment of Shock.—During the period of shock the first problem is to close the capillary network in the peripheral circulation so that no further blood plasma can escape from the vascular system. Atropine injected into the blood stream to prevent sweating, warmth applied to the body, and lowering of the head to stimulate circulation in the brain stem, as well as to

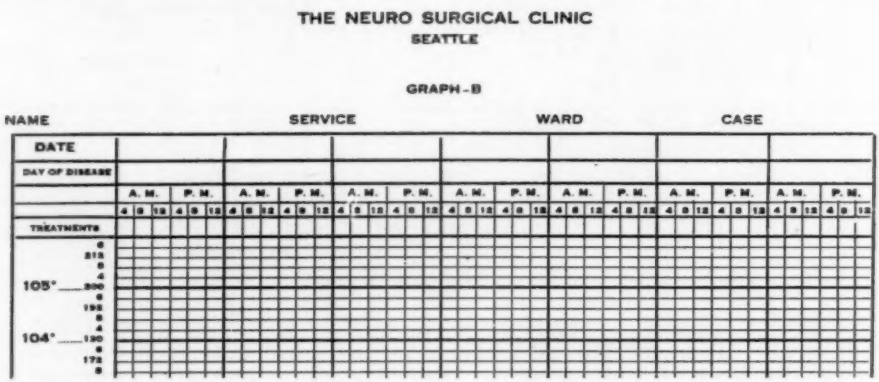


CHART II. From the Neurosurgical Clinic, Seattle. Top of four-hourly chart. The remainder of this chart, not illustrated, duplicates Chart I.

prevent secretions trickling into the lungs, are the remedies at once available. The second problem is to recapture the fluids which have left the vascular system and filled the interstitial spaces. This can be accomplished by the use of hypertonic solutions introduced into the blood vessels. The pulse pressure furnishes one clue which is very reliable during the effort to maintain this balance. The specific gravity of the urine furnishes the other valuable clue. The third problem is to preserve a constant water balance within the vascular system. Since water can escape from the skin by sweating, from the lungs during the process of respiration, from the alimentary tract by vomiting or bowel action, and directly from the blood vessels by bleeding in cases of hæmorrhage, it is essential that these four possible exits should be watched. Little can be done to control the amount of water escaping from the lungs during respiration, but since the condition of the lungs has such a marked bearing on the amount of oxygen carried to the brain, it is essential that no accumulation of mucus or fluids plug up the small bronchioles. Free oxygenation of the lungs is therefore imperative. We

use direct oxygen inhalations by means of a nasal catheter for this purpose. "Carbogen" is used at regular intervals or whenever the capillary network reveals a poor oxygen content. Vomiting, Nature's method of dehydration, perhaps causes as great a loss of water as any of the four sources of dehydration. Gamble[9] has shown how either pernicious vomiting or diarrhœa can rapidly change the blood volume and blood chemistry. The principle certainly applies in this instance. The first measure to combat vomiting is to suspend all fluid by mouth. Cerebral or medullary irritation, which after all is the cause of the gastric reaction, can be controlled sometimes by sedatives, but in bad cases only by lumbar puncture and spinal drainage.

Bleeding.—Bleeding is the fourth and most serious problem. Intracranial hæmorrhage associated with low blood pressure and pulse pressure indicates rupture of the cerebral veins and means an early death. Hæmorrhage from arterial rupture, however, soon results in signs of increased intracranial pressure and can usually be controlled, since it is dangerous only when the middle meningeal artery is ruptured.

Renal Conditions.—Renal conditions constitute a more complicated problem. Involuntary urination may mask the onset of either polyuria or oliguria. Kidney disease may cause hydration, rather than dehydration. Fay[3] has shown that this problem after all can be solved by careful attention to the intake-to-output ratio of water.

The Heart.—The heart cannot be expected to work automatically under rapidly changing conditions without some attention. True, it gives the signals when exhaustion is imminent, but aid should be given long before these distress signals are apparent. Adrenaline, pituitrin and strychnine are used to good purpose. Transfusion of whole blood is indicated when no response is obtained in combating a runaway heart.

Spinal Cord Injury.—The question of spinal injury is important. Frequently the accidents responsible for patients' injuries occur on highways, and either the patient is found stretched out on the pavement when the surgeon reaches the scene, or some passer-by has just taken him to the nearest hospital and no one can give any definite information concerning the accident. In most instances one must rely upon the neurological examination for an accurate determination of the extent of the cord damage. Frequently this examination can be made immediately without disturbing the patient, and if it is carefully carried out with little manipulation no further damage is done. Since operative interference depends upon the presence of normal reflexes and the ability to perform voluntary motions both immediately after the injury and at subsequent observations, it is important to have all information recorded. When it is impossible to get a history of the progress of neurological signs and movements of the extremities, and a positive diagnosis by skiagrams cannot be made, but a clinical examination reveals a severe spinal cord injury, an exploratory laminectomy is indicated.

The most frequent site of cord injury in association with cerebro-cranial injuries is at the second or third cervical segment; complete severance of the cord in antero-posterior dislocations is fatal within a few hours. When the lesion involves the sixth and seventh segments the upper extremities escape, and immediate extension will reduce the dislocation. These patients may live for weeks or months; therefore a routine should be established early. This consists of the use of air pads for the shoulders and buttocks, careful

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attention to the bladder, massage at stated intervals, and some mechanical apparatus for the use of the upper extremities in moving the body about in bed.

Other Injuries.—Of injuries which involve the abdominal viscera, rupture of the bladder, kidney, liver and spleen are probably the most frequent and serious. The presence of blood in the first urine specimen at once indicates a careful search for its source. If the bladder is not emptied within twelve hours it should be catheterized. Clear fluid in normal quantity relieves the surgeon of further worry; a scanty amount or the presence of blood again indicates the necessity for search as to its cause, which may possibly be a rupture along the genito-urinary pathways. Rupture of the spleen or liver is suggested whenever treatment of shock does not improve the general condition of the patient. The latter is a rare complication, but frequent enough to be kept in mind. Immediate operation, of course, is indicated. Rupture of the hollow viscera begins to cause symptoms within twelve to twenty-four hours, but does not retard the recovery from shock. Abdominal distress causes the patient to become very restless and abdominal symptoms soon appear.

Extraneous Complications.—Having made the observations detailed and established whether one or all of the complications mentioned are present, and having instituted the proper treatment for each condition, we should establish: (1) the identity of the patient, (2) the associates or witnesses to the accident, and (3) any facts which may later be of value in the reconstruction of the accident. These little observations may prove of importance, especially if the patient lies unconscious and no one has thought of material things in the confusion of the medical emergency. In addition to gathering important data, the surgeon should realize the importance of the selection of proper nurses and of issuing orders relative to visitors. Lawyers, reporters, friends and even relatives frequently cause much unnecessary trouble and harm to the patient. Demands for consultants, immediate X ray examinations, and unnecessary discussions may all be avoided if stern but intelligent management is promptly instituted. The members of the family should be informed at some length regarding these matters and their cooperation obtained.

These procedures should cover the first twelve hours during which, if the patient has not died, (1) he has recovered from immediate shock, (2) he has had hæmorrhage controlled, and (3) he has had the necessary routine established. Unless some complication now develops, the case will promptly resolve in the usual happy termination.

Summary of the Immediate Problems.—The immediate problems are those that demand immediate action: control of hæmorrhage, treatment of shock and spinal cord injuries, support of the heart and attention to other injuries and extraneous complications.

POST-TRAUMATIC TREATMENT.

After this first stage has been completed it is time to take stock and determine upon the next few days' treatment. If the surgeon is not familiar with the treatment of head injuries, he should call in expert advice at about the end of the first or second twenty-four hours. Certain examinations are

now in order and can be done safely, and the details of further care will be indicated by the results of these examinations. A more extensive general physical examination to uncover any other serious injuries is made before the patient is sent to the X ray laboratory. This examination includes an initial spinal puncture to ascertain the pressure and character of the cerebro-spinal fluid, a measurement of the output and character of the urine to determine the presence of any bladder or kidney injury, a neurological check-up, and necessary skiagrams. Cases can now be classified as: (1) simple concussion, (2) concussion with skull fracture, (3) concussion with brain damage, or (4) concussion with both skull fracture and brain damage. As the treatment varies according to the type, it is pertinent to discuss the problems presented by each type.

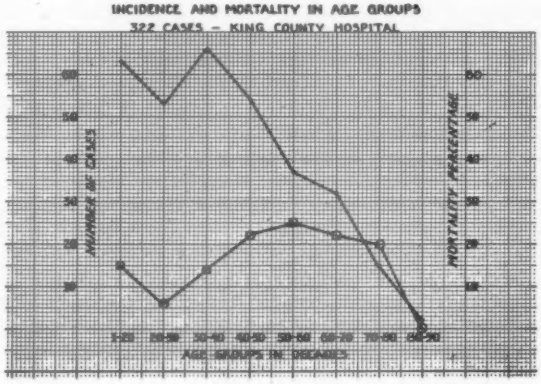
INCIDENCE AND MORTALITY IN AGE GROUPS
322 CASES - KING COUNTY HOSPITAL
NUMBER OF CASES
MORTALITY PERCENTAGE
AGE GROUPS IN DECADES

CHART III. Showing incidence and mortality in age-groups in Neurosurgical Clinic, Seattle.

Simple Concussion.—Cerebral concussion is due in our opinion chiefly to the effect of hydraulic pressure of the interstitial fluids upon the adjacent cerebral tissues (Swift,[1] Berens[8]). These effects have been described in previous papers, so they will be only briefly touched upon on this occasion. Variable factors present are: (1) the amount of cerebro-spinal fluid (interstitial) present in any space at the time of accident, and (2) the severity of the blow. If the fluid content of the brain is in a fairly normal condition, then the milder the blow, the milder the injury; the harder the blow, the more severe the damage. This is evident in the lower mortality in children and young adults and the very

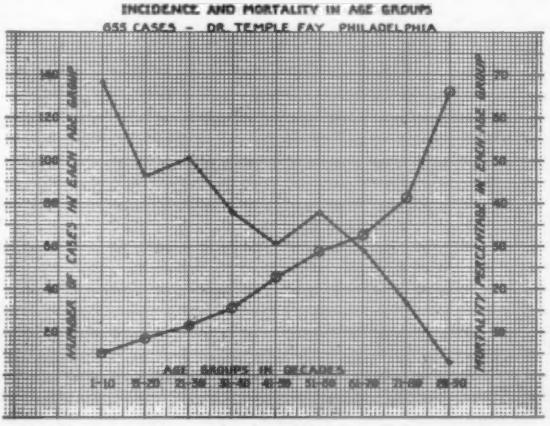
INCIDENCE AND MORTALITY IN AGE GROUPS
655 CASES - DR. TEMPLE FAY, PHILADELPHIA
NUMBER OF CASES IN EACH AGE GROUP
MORTALITY PERCENTAGE IN EACH AGE GROUP
AGE GROUPS IN DECADES

CHART IV. Showing incidence and mortality in age-groups. Clinic of Dr. Fay, Philadelphia.

high mortality in elderly patients in the group above sixty years of age. Both our tabulations and later tables by Fay show this fact to be very obvious (see Charts III and IV).

Severe concussion occurs in the group of middle-aged patients who have severe injuries and in the elderly group who have even slight injuries. Fay has shown that death is due to hydraulic pressure upon the basilar nuclei controlling the vital centres. In either group death may occur even though very little blood is present in the cerebro-spinal fluid and no fracture is found. Treatment can be directed only towards control of water balance and supportive measures for the heart. As these cases are the most hopeless ones we meet, little can be done to aid them. The mortality is low in the lower ages and rises gradually, to reach its peak in the later years.

Concussion with Skull Fracture.—In this group the presence or absence of fracture is revealed by (1) X ray examination, (2) late ecchymosis into the orbits and behind the ears, (3) discharge of cerebro-spinal fluid from the nose, (4) rigidity of the neck with excessive œdema. Depressed and compound fractures involving the vault and base of the skull complicate the picture because they may require operation. Again, the appearance of free cerebro-spinal fluid from the nares may demand operative interference to prevent rhinorrhœa with subsequent infection (Munro[10]); fracture into the mastoid process is another serious complication demanding expert care.

Concussion with Brain Tissue Damage.—This group comprises cases with bloody cerebro-spinal fluid. They are the patients who require prolonged hospitalization and develop post-traumatic neuroses and psychoses. It is our custom to give each and all of these patients repeated spinal drainage. We drain the spinal fluid every twenty-four to forty-eight hours until it is clear and the pressure normal. We regulate the amount removed by the manometric reading, which is our sole criterion as to the amount to remove. We never lower the pressure below six millimetres of mercury. With this treatment we have had very happy results.

Concussion with Both Skull Fracture and Brain Tissue Damage.—This type of lesion presents the most serious of all conditions that one sees in cerebro-cranial injuries. Spinal drainage, operative interference and post-operative complications are the factors to be considered in each case. The mortality is higher than in any group and the morbidity is largely dependent upon the response to treatment. Just when to depend upon spinal drainage, when to operate, if at all, and finally what type of operation is indicated, depend upon the nature of the case and the other circumstances. Among the latter are the advisability of transferring a patient from one place to another, perhaps the availability of surgeons and blood donors, any complicating fractures, and injuries to other parts or organs. Again, we must generalize rather than give specific methods of treatment.

Operative Interference.—The indications for operative procedures in severe head injuries are now quite generally accepted by all surgeons. All agree that certain cases require immediate operation, namely, those of middle meningeal hæmorrhage, compound comminuted fractures with profuse hæmorrhages, and œdema of the brain not relieved by spinal drainage or hypertonic solutions given intravenously. Subtemporal decompressions, radically done, are justified in hospitals where expert water balance control is not possible or at least practicable.

Spinal Drainage.—Morbidity, in our experience, means one of two conditions, œdema of the brain tissues due to actual trauma or accumulation of water in the subarachnoid or subdural spaces. If a day-by-day improvement in our patients does not appear, we at once begin to suspect one or the other of these causes. We do an initial spinal drainage, removing all the fluid that will escape, with the patient in the prone position. If this does not bring prompt improvement, the next day we instil a small amount of air and take skiagrams. These usually reveal any marked accumulation of fluid. If it is not possible to drain the accumulation, it may be necessary to operate. We use the method outlined by Penfield.[11]

The second indication for spinal drainage is bloody cerebro-spinal fluid. Blood cells accumulate in the Pacchionian filter and cause a block. Consequently fluid accumulates in the subarachnoid spaces about the Pacchionian areas and eventually a reaction takes place, both the pia and the arachnoid usually being involved. Probably a biochemical reaction, due to the by-products of traumatized brain tissue, plays some part in this picture. We believe that frequent and thorough spinal drainage will prevent this and also reduce the morbidity (see Table II).

TABLE II.

Morbidity in Spinal Drainage Cases as Compared with that where Spinal Drainage was not Carried Out. Series A had spinal drainage in all cases with bloody cerebro-spinal fluid. Other groups did not have spinal drainage. Group B had spinal punctures after twenty-four hours for diagnostic purposes.

	Number of Cases.	Less than One Week.	One to Two Weeks.	More than Two Weeks.
King County Hospital—Series A	145	107 73·0%	28 19·0%	10 6·9%
Series B	169	130 77·0%	25 15·0%	14 8·3%
Total	314	237 75·0%	53 17·0%	24 7·0%
Other Seattle hospitals ..	222	144 65·0%	40 18·0%	38 17·0%
All hospitals in Washington	1,433	960 67·0%	249 17·0%	224 15·0%

Dehydration.—Dehydration as understood in the treatment of head injuries means the removal of water from the body. This is automatically accomplished by certain physiological reactions, namely, breathing, sweating, vomiting, elimination of the urine and action of the bowels. Artificial dehydration is carried out either by elimination of water from the vascular system through the alimentary tract or by spinal drainage. The most common method is by the instillation of concentrated magnesium sulphate solution in the lower bowel. The fluid must first be returned to the vascular system

before it can be taken from the bowel. For this purpose hypertonic solutions are given intravenously, and after the fluid has entered the vascular tree it may be brought to the bowel by the concentrated enema and finally removed from the body. Many do not understand this double change of fluid volume and think that it is necessary to remove the fluid from the body in the stage of shock. Frequently one sees cases in which repeated glucose injections followed by copious administration of magnesium sulphate enemata have so dehydrated the patient that headache and almost stupor have developed. A good plan to follow is not to use magnesium sulphate during the first twenty-four-hour period, since dehydration is contraindicated during the period of shock. After the period of shock has passed, it is essential to keep the water balance at a constant level and dehydration may be indicated. Control of water intake and output, variation of the amounts, and finally readjustment of the body to the changed conditions, require expert and constant attention on the part of the surgeon. Fay's chart, which we have modified, gives a graphic picture of the day-by-day water balance. The blood pressure, urine concentration and pulse pressure are indicative of the condition of the water balance.

Post-Traumatic Neurosis.—One of the complications of a severe cerebro-cranial injury is the so-called post-traumatic neurosis. To prevent this the surgeon should be on the alert for the early symptoms. Patients begin to brood over their injuries, possibly realizing that they do not function properly either mentally or physically. Soon suggestions, offered from the outside, so substantiate this idea that what was at first merely a suggestion or thought, gradually becomes a real belief. It is but a short step from this to the typical neurosis, that is, loss of independence and self-assertion, inability of the patient to assume his former status in society, and a complete failure to readjust himself to his present environment. More functional symptoms, headache, generalized bodily weakness, sweating, fainting, loss of appetite and consequent loss of body weight, follow. Unless checked, these patients become hopeless invalids and are eventually a total economic loss.

How can these dire results be prevented? We believe that the surgeon's duty is to use every means at his command. The first, and probably the most important, factor involved is the environment of the patient. Only those who are cheerful, intelligent and strong physically should be permitted to be near the patient or to have any contact with him. The subject of the injury should not be discussed until the patient has recovered from all of his actual symptoms. The injured parts should be considered as not seriously harmed, and as soon as the mental condition of the patient is such as to retain any facts, assurance of rapid recovery should be given. The greatest problem is that of the family and friends. An early explanation of these conditions and the effect upon the patient's future will usually break down any opposition and enlist their support. In cases where cooperation is not obtainable, the patient must be protected by the only other remedy, that is, sedatives. The second factor is one of medico-legal character: the responsibility for the accident, compensation and possible criminal responsibility. This problem is more difficult. Each case presents its own points and must be so treated. Generally, one need not worry much over this type of neurosis. After the court has had its say, symptoms usually disappear.

Traumatic Psychosis.—Psychoses are traumatic, not post-traumatic. When the patient does not awaken to normal mental reactions and responsibilities, but develops a psychotic pattern, we have an entirely different picture from that which develops in post-traumatic neuroses. Traumatized brain tissue, especially in severe concussion, with or without bloody cerebro-spinal fluid, is very likely to cause psychoses. These are present in the patients with delusions, hallucinations, maniacal outbursts, severe vertigo and unstable physical responses. Care, different from that required for the neuroses, is necessary. Restraint, prolonged use of sedatives, and sanatorium treatment, not for days but for months, are frequently necessary. Changes of personality, sometimes permanent, and homicidal and suicidal tendencies must be detected and measures taken to prevent serious outbursts.

SUMMARY OF POST-TRAUMATIC TREATMENT.

The following are the problems which demand consideration in post-traumatic treatment:

1. Classification of cases as those of (*a*) simple concussion, (*b*) concussion with skull fractures, (*c*) concussion with brain tissue damage, (*d*) concussion with skull fracture and brain tissue damage.
2. Operative interference.
3. Dehydration.
4. Spinal drainage.
5. Post-traumatic neurosis.
6. Traumatic psychosis.

CONCLUSION.

We have shown that the care of a severe cerebro-cranial injury naturally falls into two periods, the first of which is that of the immediate treatment which covers the first twenty-four- to forty-eight-hour period. This treatment is usually directed by the doctor first called to the patient and is fairly well standardized. The second period is that of the post-traumatic treatment, which begins about the second day and extends from a few days even to months. This should be directed by an experienced surgeon, as the treatment varies with the character of the injury, the complications due to associated injuries, and the reactions due to damage of brain tissue. By following these practices, we have found a marked decrease in the morbidity and mortality following severe cerebro-cranial injuries.

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DRAINAGE IN ACUTE APPENDICITIS.

By Sir Alan Newton,
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I have been impressed, in the course of my consulting work in recent years, by the attitude of doctors towards the use of a drainage tube in the treatment of acute appendicitis. Many appear to believe that it is tantamount to a confession of surgical incompetence to drain the abdomen after the removal of an acutely inflamed appendix, that the mere presence of a tube in the wound brings in its train dangers greater almost than those attending the inflammation of the appendix itself, and that if a drainage tube be used, it should be removed not later than forty-eight hours after the operation. As a result of this widespread belief, I have seen in consultation many patients, in the last few years, in whom death or prolonged post-operative morbidity, after the removal of an acutely inflamed appendix, was attributable, in my opinion, either to lack of drainage or to premature removal of the tube. It therefore seems to me desirable that the question of drainage in acute appendicitis should be reconsidered. Such a discussion is rendered all the more necessary by the facts that the treatment of acute appendicitis is today the task of the great majority of members of our profession, that many of these men must rely upon what they have read in print rather than upon what they have experienced in practice, and that, in some recent publications in surgical literature, the drainage tube has been impugned.

Thirteen years ago, St. Leger Brockman[1] advocated a more restricted use of the drainage tube in cases of acute appendicitis, and pointed out that, though in many cases the advantages of drainage were obvious, there were a great number in which its sole value was its hypnotic influence upon the surgeon. In his opinion it was unnecessary to employ drainage in many cases, even when the appendix was gangrenous or had perforated, providing that the disease had been present for less than three days, the patient was over twelve years of age, the degree of toxæmia was not excessive, cyanosis was not present, and there was not a large area of raw tissue left after the removal of the appendix. He was also guided by an immediate examination of the peritoneal exudate, and believed that drainage was required when this revealed a lack of large mononuclear cells, failing power of these of absorbing stains and an absence of phagocytosis. Having adopted these precautions against dispensing with the use of drainage, he was able to report a decreased incidence of fæcal fistula and of intestinal obstruction due to adhesions and a complete immunity from residual abscess in undrained cases.

Four years ago, A. J. Trinca[2] advocated the abolition of the drainage tube whenever possible in the treatment of peritonitis, of which acute appendicitis, as he pointed out, was the most common cause. He stated that attempts at drainage of the peritoneal cavity were futile and might

be productive of great harm, and attributed a large number of post-operative complications to the presence of a drainage tube.

There is no doubt that these and other similar papers have had a widespread effect, not only because the authors have been able to report creditable mortality and morbidity figures, but also because surgeons generally are distressed by the fact that there has been no appreciable decrease in the mortality from appendicitis in recent years and are therefore anxious to revise their operative procedures. This effect, in my opinion, has been most harmful. Six years ago, in a review of 995 cases of acute appendicitis treated at the Royal Melbourne Hospital,[3] I pointed out that, though the dictum "when in doubt, drain" was generally obeyed, a tube having been used in 38·4% of the total number of cases, this policy was temporarily abandoned after the publication of Brockman's paper. The results were immediate and catastrophic. There ensued two cases of residual abscess, in one of which the patient died; peritonitis with ileus in another case, in which the patient died, and fatal pylephlebitis in a fourth case. Though it is possible that these results would not have been improved by the use of a drainage tube, there seems to me to be little doubt that the surgeons concerned would have felt happier had they enjoyed the "hypnotic influence" of its presence. It is also possible that these surgeons failed to pay due attention to the precautions against dispensing with drainage which Brockman was careful to supply, because it is a very human failing to retain in the memory the general impression conveyed by a paper and to forget the qualifications and restrictions which the author has emphasized. The general impression conveyed by Trinca's paper is that drainage is futile, although a critical study reveals that the author actually advises this procedure in the more advanced cases of appendicitis, such as those in which, owing to technical difficulties, it is impossible to remove the appendix in whole or in part, those in which a fæcolith or gangrenous *débris* has escaped, or those in which a raw area is left after appendectomy. I confess that I am at a loss to explain why he should advocate the use of a "futile and possibly harmful" procedure in those patients who are already sufficiently handicapped by the severity of their condition. I am forced to conclude that Trinca, at heart, is an ardent advocate of drainage in suitable cases, and will be the first to lament the general impression that he opposes the use of a drainage tube.

A potent factor in restricting the use of a drainage tube is the fear of possible sequelæ, such as fæcal fistula, residual abscess and intestinal obstruction. These risks have been exaggerated. In the Royal Melbourne Hospital series of 995 cases drainage was used in 382. There were fifteen cases of fæcal fistula, two of which occurred in patients not treated by drainage. The incidence after drainage was 1·3%. Thirteen of the fifteen cases healed spontaneously. There were seventeen cases of residual abscess, in two of which drainage had not been employed, so that the incidence after drainage was 1·5%. The incidence of intestinal obstruction during convalescence was only 0·5%. In order to supplement these figures I have investigated the results of the treatment of acute appendicitis in my private practice for ten years prior to December, 1936. I do not see many cases of this disease, as is shown by the fact that during this period the total number was 110, though during the same time I performed 589 operations for stones in the biliary tract. There were two deaths in the 110 cases, one

patient being a child who died from an associated ulcerative colitis and the other a man of seventy-two years of age. Drainage was employed in 43 cases, in none of which fæcal fistula, residual abscess or intestinal obstruction developed. There was one case of incisional hernia in a man aged eighty-three, and a fatal case of ileus associated with general peritonitis in the man aged seventy-two. On the other hand, there was one case of fæcal fistula associated with incisional hernia in a case not treated by drainage, and another not treated by drainage died of intestinal obstruction due to an adhesion twelve months after appendicectomy. Severe post-operative ileus developed in one case where drainage was not carried out, but recovery followed appropriate treatment carried out by Dr. Ian Wood. These figures suggest that the risks attending the use of a drainage tube have been greatly exaggerated and, further, that complications usually ascribed to the presence of a tube occur not infrequently when no drainage has been employed. I have never regretted the presence of a tube, but more than once I have bitterly lamented its absence.

It is my practice to operate, in the great majority of cases of acute appendicitis, through a muscle-splitting incision, and to drain, when necessary, through this incision. When a drainage tube is used it is necessary to guard against the effects of pressure exerted by the tube upon the structures in its vicinity. This can be achieved by replacing the tube, after forty-eight hours, by another of equal length but smaller diameter. This tube is replaced, after a similar time, by a third, which is of about the diameter of bicycle valve tubing, and which is finally removed when the condition of the patient warrants it. The practice of progressive shortening of the original tube is undesirable, in my opinion, because it fails to obviate the effect of lateral pressure, and may be followed by the development of a residual abscess in the lower part of the track from which the tube has been withdrawn by the shortening process. The use of tubes of diminishing diameter, but of similar length, has the additional advantage that the wound contracts down progressively upon the tube track, thus obviating the risk of an incisional hernia. This contracting process is assisted by the fact that widespread wound infection is much less common when drainage is employed.

In conclusion, I have, I hope, made it clear that, in my opinion, complications usually ascribed to the presence of a tube are not uncommon when drainage has not been employed, and that drainage can be used in acute appendicitis without fear that its disadvantages will outweigh the undoubted advantages which accrue from its use. May I therefore suggest that there should be a general reversion to the practice best expressed by the axiom "when in doubt, drain", and that surgeons should not hesitate to use a tube in any case in which the condition of the operative field is not entirely satisfactory?

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ACHALASIA OF THE ŒSOPHAGUS.

By Richard Flynn,
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Introduction.

Achalasia of the œsophagus or cardiospasm ranks next in frequency to carcinoma as a cause of œsophageal obstruction. This fact is apparently not fully appreciated, and cases of cardiospasm are not only frequently not recognized, but are also wrongly diagnosed and treated. The occurrence of several cases in my own practice has led me to review the subject and to present a series of case histories which illustrate very well some of the associated problems.

In this lesion the cardiac sphincter does not relax in the act of deglutition and the food is retained in the lower end of the œsophagus, the peristalsis of which is not sufficiently strong to overcome the resistance offered by the unrelaxed sphincter. The resistance of the unrelaxed cardia can only be overcome by a pressure equal to about twenty centimetres (eight inches) of water. Consequently when a meal is taken, no food passes into the stomach, in spite of violent but intermittent peristalsis, until the contents of the œsophagus form a column about twenty centimetres (eight inches) in height. Then when something further is eaten or drunk the cardia opens and allows food to pass till the height of the column is reduced to twenty centimetres (eight inches), when it closes once more.

Ætiology.

Attention was first drawn to the syndrome by Purton in 1821, and the first series of cases was reported by von Ziemssen and Zenkin in 1878. Their report was based mainly on *post mortem* findings, and they added little to the clinical knowledge of the condition. The writings of Sippey, Hurst, Plummer, Vinson and Moersch have drawn the attention of British and American practitioners to the importance of this clinical entity and to its symptomatology, pathology and treatment.

The question of the ætiology of cardiospasm has long been debated. Many of the older physicians regarded it as a purely functional disorder; in fact, one of the patients (Case II) whose history is given later, was advised by a distinguished colleague to be patient and that everything would be well after the menopause! Others argue that because it is rarely met with in children it is purely functional, but the series of cases reviewed by Moersch[1] shows that this argument is unsound, as in three of his cases the symptoms commenced during the first year of life.

According to Plummer,[2] the disease has been attributed to the following causes: primary cardiospasm (Meltzer); primary atony of the musculature

of the œsophagus (Rosenheim); simultaneous presence of cardiospasm and paralysis of the circular fibres of the œsophagus brought about by paralysis of the vagus (Kraus); congenital disposition (Fleiner, Zenker, Luschker and Seevers); primary œsophagitis (Martin); and kinking at the *hiatus œsophagi*. Plummer himself in his early papers favoured the theory of primary spasm of the cardia. Hill and Johnson both suggested that the fault lay in the muscle bundles of the diaphragm surrounding the œsophageal hiatus, the former believing that there was an absence of the normal relaxation of the diaphragm during swallowing, and the latter that there was an actual spasm of its fibres, that is, a phrenospasm. As, however, the dilatation of the œsophagus often extends through the diaphragm, both of these theories can be discarded. Another view is that the dilatation of the œsophagus is due to the pressure of the liver on the abdominal portion of the œsophagus. Lockwood,[3] in 1903, suggested that cardiospasm might be due to a reflex contraction of the cardiac orifice owing to interference with the vagus. In the case he reported he suggested that the reflex was set in action by the irritation set up owing to the dislocation of the right kidney by a fall.

Einhorn in 1888 came to the conclusion that he was dealing with a "lack in the reflex relaxation or opening of the cardia during the act of swallowing". He pointed out that Kronecker and Meltzer had shown that "every act of swallowing easily opens the cardia by reflex action". He then suggested that:

> If the centre of, or the circuit of, this reflex action is in any way disturbed so that the cardia does not relax with swallowing, there will necessarily result a slight difficulty in transferring food from the œsophagus into the stomach.

Rolleston (1896) arrived at the same conclusion independently, and Hurst (1913) made a similar suggestion in discussing a case reported by Parkes Weber.[4] In the course of this discussion he said that after examining the specimens of so-called cardiospasm in the museum of Guy's Hospital, he had come to the conclusion that the condition could not arise from a spasm of the cardiac sphincter, which was then the commonly accepted view. As the symptoms were often present for many years before death, it was quite inconceivable that a spasm of such long duration should not lead to any hypertrophy of the cardiac sphincter. Yet in all the specimens that he had examined the absence of hypertrophy of the cardia and the absence of any obvious obstruction after death had been in striking contrast with the degree of hypertrophy of the œsophageal wall. As a result of this study he came to the conclusion that that condition was really due to the absence of the normal relaxation which should occur when each peristaltic wave travelling down the œsophagus reached the cardiac sphincter, or, in other words, to an incoordination of the neuromuscular mechanism of the lower end of the œsophagus. Parkes Weber suggested that it might be more of a dissociation than an incoordination.

As a result of this incoordination or dissociation the œsophagus becomes atonic, dilated and elongated, reaching a maximum diameter immediately above the diaphragm, or it may become S-shaped with the dilated portion lying over the upper surface of the right cupola of the diaphragm. The dilatation generally extends as high as the third dorsal vertebra. Wakeley[5] stated that the reason for the œsophagus's dilating towards the right was that the aorta lay to the left, and being a resisting structure, prevented any dilatation towards the left side. Whiteford,[6] in reporting a case, stated that

the œsophagus above the diaphragm was dilated throughout to a diameter of five centimetres (two inches).

Acting on Hurst's suggestion, Rake,[7] of Guy's Hospital, demonstrated in six cases well marked inflammatory and degenerative changes in the lower end of the œsophagus, particularly involving Auerbach's plexus. Stokes in two other cases found that the whole of the nerve plexus in the neighbourhood of the cardiac sphincter had been destroyed and replaced by fibrous tissue. Hurst[8] (1932) stated that achalasia of the œsophagus was the only malady known definitely to be due to organic disease of the sympathetic nervous system, adding that eighteen cases had been recorded up till then of necropsies performed on patients with achalasia, in which inflammation or degeneration of Auerbach's plexus in the cardiac sphincter had been found. In accordance with this view, he had found that in spite of the fact that peristaltic waves were unable to overcome the obstruction, the weight of a rubber tube filled with mercury was sufficient to enable it to drop without difficulty through the unrelaxed cardia into the stomach; and that it could be withdrawn without difficulty, whereas it would be tightly grasped by the sphincter if spasm were really present. To express in the name of the condition this change of view as regards its ætiology, he suggested that the term "cardiospasm" should be replaced by that of "achalasia", a term suggested by Sir Cooper Perry.

Reid, in 1839, had noted that in rabbits vagal section in the neck caused stasis of food in the œsophagus. McKenzie also stated that when the vagi were stimulated the œsophagus contracted and the cardia opened, and when the vagi were paralysed the œsophagus dilated and the cardia closed. Lockwood[3] (1903) had suggested that cardiospasm was due to a reflex contraction of the cardia owing to interference with the vagus.

The nerve supply to the œsophagus is still a matter of controversy. Gaskill states that it receives no sympathetic fibres, whilst others state that it receives sympathetic fibres throughout its whole course. It is possible that the truth lies between these two extreme views, and that the cardiac sphincter has a dual nerve supply from the vagi and sympathetic which would be expected to be mutually antagonistic in action. Since Hunter and Royle drew attention to the importance of the sympathetic system from a surgical viewpoint, many workers have investigated the function of the sympathetic supply to the cardia.

Ross and Gask,[9] in discussing achalasia, said:

> It has been suggested that this condition may be amenable to treatment by sympathectomy. Before such treatment can be placed on a rational basis more information is required with regard to the anatomy of the sympathetic supply to the œsophagus and the rôle which the sympathetic plays in deglutition. It will also be necessary to define more clearly the site and the nature of the pathological process in cardiospasm, and should it be established, as it is commonly supposed, that an inflammatory or degenerative lesion of Auerbach's plexus is invariably present, division of the sympathetic supply proximal to Auerbach's plexus would not be expected to be beneficial. These problems involved in the proposal to treat cardiospasm by sympathectomy are in urgent need of solution.

The most recent, and in many respects the most complete, study has been published by Knight.[10] He has reached the following conclusions:

1. The œsophagus receives a sympathetic innervation.
2. There is a true intrinsic sphincter at the cardia.

3. Vagal stimulation causes tetanic contraction of the upper third of the œsophagus, which is composed of striped muscle. This contraction is augmented by sympathetic stimulation.

4. Vagal stimulation causes increased tonus and mobility of the lower third of the œsophagus, which is composed of plain muscle. The tonus and mobility are inhibited by sympathetic stimulation.

5. Bilateral vagal section, if complete, reproduces the condition of achalasia of the cardia. Simultaneous removal of the sympathetic fibres prevents the onset of this obstruction. When the obstruction develops it can be relieved by section of the sympathetic supply to the sphincter.

6. The sympathetic supply of the cardiac sphincter is accessible as it passes to the sphincter along the course of the cœliac axis and left gastric arteries.

Occasionally achalasia is the result of some organic disease, such as tabes or peripheral neuritis; occasionally it is reflex and secondary to irritation of the œsophagus by adhesion to a tuberculous gland or to an ulcer or growth of the stomach (Case IX). In the large majority of cases no obvious cause is found; the condition is then due to inflammation, degeneration or syphilis involving the nerve plexus of the cardia, and may be regarded as analogous to the local disease which gives rise to heart block.

CLINICAL FEATURES.

The disease occurs in patients of all ages and is equally common in both sexes. Moersch[1] (1929) reported that thirty-four out of six hundred and ninety-one patients suffering from achalasia of the œsophagus, whose histories he reviewed, placed the period of onset of their symptoms before the age of fourteen years, and of these, twelve were brought for consultation before they had reached that age.

Langmead[11] reported the history of a female child, sixteen months old, who commenced to retch and regurgitate her food soon after birth. Parkes Weber[12] collected records of eleven other cases occurring in the first seven years of life, the youngest being a patient of Chevalier Jackson,[13] a baby two days old. In a series of twenty-three cases reported by Hurst,[7] twelve were in men and eleven in women. The age of onset varied between twenty-one and seventy-one years, and in seven of his series it was before the thirtieth year. The average age at onset in Plummer's series was twenty-nine years.

Plummer has stated that "the symptom complex in cardiospasm is as a rule almost pathognomonic". The course of the disease may be divided into three stages:

1. Achalasia without regurgitation of food, characterized clinically by a complaint of discomfort behind the lower end of the sternum.

2. Achalasia with immediate regurgitation of food.

3. Achalasia with dilatation of the œsophagus, retention of food in the œsophagus and its regurgitation at irregular intervals after ingestion.

The onset of achalasia is usually insidious, though Hurst states that it may be quite sudden, the patient as a rule noticing equal difficulty in swallowing fluids and solids. The dysphagia may be intermittent, and is frequently associated with regurgitation of food, which is usually in the same condition as when it was swallowed, not mixed with gastric juice, but commonly containing much mucus secreted by the œsophageal mucosa. If the

food is retained in the œsophagus sufficiently long it may undergo fermentation.

Pain is a variable symptom. When present it is frequently sharp and is felt under the lower end of the sternum, but it may radiate into the back or up into the neck. It is especially noticeable when food passes through the cardia into the stomach. The severity of the pain varies from a slight discomfort to a degree severe enough to cause the patient to double up, in which case gall-stones may be considered to be the cause (see Case III). At times the patient complains of a feeling of tightness and great discomfort in the chest after meals until the food is rejected by ejection. Vomiting does not take place, as the stomach takes no part in the action, which is, moreover, unaccompanied by nausea. Hurst and Rake[7] state that the ejection is generally a more or less voluntary action which the patient learns to perform whenever the substernal sensation becomes sufficiently unpleasant. Regurgitation rarely occurs on lying down, although the œsophagus is filled with a large quantity of food and mucus, because the upper end of the œsophagus is kept closed by the sphincteric action of the crico-pharyngeus muscle. The duration of the symptoms varies greatly in different cases; some patients have to seek relief almost immediately after their onset, while others with milder symptoms may suffer almost a lifetime before seeking medical relief.

The average patient soon learns to take several swallows of solid food and then to force the food through the cardia by rapidly drinking a glass of water, and so uses the voluntary muscles of the pharynx to overcome the delay at the cardia, employing, in fact, a natural hydrostatic dilator. Vinson,[14] by study of moving picture films of a patient with cardiospasm, showed that the patient also used the voluntary muscles of the neck to force the chin downwards and forwards on the neck, and so to increase intrathoracic pressure which assisted in overcoming the obstruction.

Food is propelled from the dilated œsophagus into the stomach by the forces of gravity and increased intraœsophageal pressure. The force exerted by gravity equals the height of the column of liquid in the œsophagus. The intraœsophageal pressure is raised above normal by those forces which raise intrathoracic pressure and by the crowding of more food into the partially filled œsophagus. At each act of deglutition sufficient food is forced into the stomach to lower the intraœsophageal pressure to a point where it is no longer sufficient to overcome the resistance offered by the cardiac sphincter.

An associated clinical finding, which is almost constant, is hypertrophy of the salivary glands, especially of the submaxillary glands. Owing to inspiration into the bronchial tubes of regurgitated food, bronchiectasis and pulmonary abscess may occur as a complication.[15] Spontaneous pneumothorax has been reported by Vinson,[16] who also reports the cases of two patients with symptoms and findings typical of achalasia, in each of whom a diverticulum of the middle third of the œsophagus was found complicating the achalasia.[17] The cause of these diverticula, he stated, was purely speculative; they might have been primarily pulsion diverticula, due to pressure on a congenitally weak wall, or they might have been tiny traction diverticula with secondary enlargement on account of obstruction at the cardia. Hurst (1928) also described a single case of achalasia complicated by diverticulum.

DIAGNOSIS.

A diagnosis of cardiospasm must not be made unless there is direct radiographic evidence of obstruction at the cardia. Psychoneurotic individuals frequently complain of a sense of obstruction to food in the chest, without radiographic evidence of obstruction. Dilatation of the cardia of these patients is useless unless attention is paid to the other factors in the neurosis.

Features which should suggest a diagnosis of achalasia are: (*a*) dysphagia existing over a lengthy period without increase of symptoms, (*b*) the fact that the patient experiences as much difficulty with fluids as with solids, (*c*) the absence, on appropriate testing, of gastric juice from the ejected food. These features are strongly suggestive, and should lead to a radiographic examination of the œsophagus. If achalasia is present, the skiagram will show the lower end of the œsophagus ending in a perfectly smooth funnel, which has been likened to a cigar-tip. The passage of a number 45 Charrière olivary-tipped tube over a previously swallowed silk thread, without more than a slight resistance at the cardia, is confirmatory evidence.

Occult blood may be found in the stools in carcinoma of the œsophagus, but never in achalasia. Œsophagoscopy is unnecessary, as the diagnosis can be made with more certainty and with much less discomfort by means of radiography. The passage of an ordinary bougie, unless along a swallowed thread, is dangerous as well as useless, for, as mentioned previously, the lower end of the œsophagus may be so dilated that it forms a blind ending, with the convexity to the right over the diaphragm, and in addition is often weakened by the presence of an œsophagitis. Even with the utmost care the bougie may be pushed through the œsophageal wall and so give rise to infective complications, such as mediastinitis.

DIFFERENTIAL DIAGNOSIS.

The complaint of "difficulty in swallowing" or "that food is regurgitated" calls for a thorough investigation by the clinician. The means at his disposal are a detailed history and physical examination, the passage of œsophageal bougies over a previously swallowed silk thread, œsophagoscopic examination and radiographic investigation.

The causes of dysphagia are: (1) a tumour pressing on the œsophagus from the outside, for example, an aneurysm of the thoracic aorta or an enlarged, possibly malignant, thyreoid gland; (2) carcinoma of the œsophagus; (3) stricture of the œsophagus; (4) foreign body in the œsophagus; (5) pharyngeal diverticulum; (6) diverticulum of the lower third of the œsophagus; (7) the Plummer-Vinson syndrome; (8) paralysis of the œsophagus or pharynx, which is more frequent, due to disease of the central nervous system, for example, bulbar palsy or hæmorrhage into the pons; (9) interference with the intrinsic nerve supply of the œsophagus by involvement in inflammatory swelling or newgrowth; (10) *myasthenia gravis*; (11) recurrent inclusion of the stomach in the sac of a diaphragmatic hernia; (12) hysteria; (13) cancer of the cardiac end of the stomach; and (14) achalasia of the cardia.

Hysterical dysphagia is relatively infrequently met with, and before such a diagnosis is made all organic causes must be excluded. The history

and physical examination will be found particularly helpful in diagnosing the lesions numbered (1), (8) and (10) in the above list, and frequently are all that is necessary to diagnose pharyngeal diverticulum. The history alone is often sufficient to diagnose cancer, cardiospasm and stricture.

Cancer of the œsophagus is the most frequent cause of dysphagia; it is more common in men than in women, and the history of progressive difficulty, first in swallowing solids, then with semi-solids and finally with fluids, is almost diagnostic. Radiographic evidence is helpful, while œsophagoscopic examination with the taking of a biopsy leads to a final diagnosis of malignancy and its degree by the pathologist.

Cicatricial stricture following ingestion of caustics, accidentally by children or by adults with suicidal intent, is generally diagnosed provisionally from the history, and the diagnosis is confirmed by radiographic examination. The condition is treated by the passage of bougies over a previously swallowed silk thread. Strictures are occasionally congenital, and Vinson has reported one case due to the tubercle bacillus. They sometimes follow as a complication of typhoid fever and severe vomiting of pregnancy.

Œsophagoscopic and radiographic investigation are generally needed for the diagnosis and treatment of foreign body in the œsophagus, and for the diagnosis of œsophageal diverticulum.

The dysphagia of the Plummer-Vinson syndrome is always associated with a hypochromic anæmia and frequently with a well-marked atrophic glossitis and achlorhydria. The syndrome is most frequently met with in females between fifteen and fifty years of age, who are generally quite thin and edentulous, and are often the possessors of the so-called spoon-shaped nails. The cause of the dysphagia in this syndrome is thought to be a similar atrophic change in the pharyngeal and œsophageal mucous membrane to that observed in the mucous membrane of the tongue. A blood count will clear up the diagnosis, and large doses of iron will usually cure the condition. Ahlbom[18] has drawn attention to the frequency with which this syndrome, if neglected, is associated with carcinoma of the mouth and œsophagus.

Carcinoma of the cardiac end of the stomach is sometimes difficult to diagnose from cardiospasm, and Case IX illustrates this difficulty and a method of surmounting it.

TREATMENT.

Russell[19] (1898) was the first to suggest the use of an expansible dilator. He describes his dilator as follows:

> I accordingly made an instrument consisting of a sausage-shaped silk bag rendered airtight by a very thin rubber bag within, and mounted on the end of a tube or hollow syringe. This was passed through the stricture in a collapsed state and blown up by an air syringe when in position.

Lockwood[3] (1903) used a rubber bag which was distended by air pumped in by an ordinary hand pump. The distension of the cardia was repeated three times a day before meals, till the bag broke. He reports that the patient obtained relief and gained 6·3 kilograms (fourteen pounds) in weight. Seeward Marsh recently demonstrated at a meeting of the Royal Australasian College of Surgeons a patient similarly treated with relief. Larimore[20] (1917) reported that he had found air inflation of the balloon most satisfactory. He used the pump from a sphygmomanometer to inflate

the bag, but found that the manometer need not be used, as the expression of pain by the patient was an adequate and sure guide. He did not use a manometer or a silk thread.

In 1903 Dunham[21] described his method of dilating a stenosed œsophagus by making the patient swallow a thread which was withdrawn from the stomach through a gastrostomy opening. It was then used for drawing down spindle-shaped bougies through the stricture. In 1909 Mixter modified Dunham's method by allowing the thread to travel into the intestine sufficiently far to allow of its being drawn taut, thus eliminating the need for a gastrostomy.

Plummer points out that in passing instruments through a narrowed œsophagus the two roles of safety are: (*a*) the tip of the instrument must be constantly under the guidance of vision, or (*b*) the instrument must follow a guide in such a way as to make perforation of the œsophageal wall impossible. Hurst[22] uses rubber tubes varying in diameter between 1·309 and 1·825 centimetres ($\frac{33}{64}$ and $\frac{46}{64}$ of an inch) (gauge numbers 28 to 34), 77·5 centimetres (31 inches) long, and each containing the same amount of mercury 595·3 grammes (one pound five ounces), which he has found by experience to be sufficient to force open the closed sphincter. He states that it is best to pass the bougies on the first occasion behind the fluorescent screen; it is then seen that they pass without deflection into the stomach. When the lower 7·5 centimetres (three inches) of the tube are within the stomach, a mark is made on the tube at the level of the incisor teeth. Larger tubes are passed at a single sitting, and in most cases the largest tube meets with no more resistance than the smallest, which is another proof of the absence of spasm in this condition.

Hurst then instructs the patient to pass the largest tube on himself before each meal and to leave it in as near a quarter of an hour as possible. Gradually the frequency of the passage of the tube is reduced until it is finally discontinued. The patient retains the tube so that he can pass it if at any time he feels that some slight obstruction has returned. Vinson, using Plummer's method of dilatation, claims that 75% of sufferers from achalasia are relieved by a single dilatation, and that in the other 25% there is likely to be a return of trouble within a year, and that in these relief is obtained by further dilatation.

The treatment adopted in three of the cases reported in this article was that recommended by Plummer. The other five patients were treated by dilatation with the Moersch dilator. In Plummer's method the patient is given a twisted silk thread to swallow and is instructed to swallow about 5·49 metres (six yards) of the thread slowly, not faster than 30 centimetres (one foot) per hour. If the thread is swallowed faster than this, or the patient chews it, snarls and knots are produced which render the thread useless for passing the dilator. The patient is permitted to have his usual meals while he is swallowing the thread, and he sleeps with the thread in place.

According to Plummer, the thread has a twofold purpose: (*a*) it acts as a most efficient guide when bougies are being passed through the cardia, somewhat similar to the wires in the old-fashioned cash carriers in our

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large shops, (*b*) when the thread is drawn tight it prevents the operator from pushing a hole through the wall of the œsophagus when he is passing bougies. The use of the thread is also invaluable in tight œsophageal strictures. If a skiagram of the dilated and tortuous œsophagus in a case of achalasia is studied, it is easily understood how valuable the use of the thread is. After the thread has been swallowed it passes through the stomach into the duodenum and on into the small intestine, and it becomes so firmly anchored that it tends to break rather than to come out when it is pulled upon.

The time necessary for the average patient to swallow sufficient length of thread is about twelve hours. I have found that a small slotted lead shot, as suggested by Moersch, is sometimes helpful in getting the thread started, and it also allows the end of the thread to be visualized radiographically.

No form of anæsthesia must be used, since the discomfort of dilatation serves as an indication of the amount of dilatation necessary. If the patient does not experience some discomfort behind the lower end of the sternum during the dilatation, it means as a rule that the cardia has not been sufficiently dilated. Again, any form of general anæsthesia exposes the patient to the risk of inspiration into the bronchial tree of mucus or food retained in the œsophagus, with the risk of pneumonia, bronchiectasis or lung abscess.

Once the thread is fixed, a distensible silk-covered rubber bag is passed and is dilated with water to a pressure not greater than 5·58 metres (18·24 feet) of water. Dilatation is generally accompanied by substernal discomfort. In 1926 Vinson and Moersch commenced to use a 60 Charrière bougie to dilate the cardiac orifice before using the silk bag. They did this to diminish the risk of rupturing the œsophagus, and they frequently found that dilatation with this bougie was sufficient to relieve the condition. Five of the cases here reported have been so treated with satisfactory results.

Apart from dilatation methods, the following operative procedures have been advocated from time to time.

Russell mentioned that he had seen Loretto, of Bologna, stretch the cardia from below in cases of cardiospasm, and suggested that this might be tried when dilatation from above failed.

Mickuliez (1904) reported four cases in which he had dilated the cardia from below with good results. Besides Mickuliez, Erdman (1906), Judd, Vinson and Greenlee[23] (1929), and Butler[24] (1931) reported cases relieved by this method. Judd emphasized the value of the swallowed silk thread in locating the cardia when dilatation was carried out from below. Walton[25] also warmly advocated the retrograde method and reported excellent results in thirteen out of fourteen cases treated by him. Exner (1917) performed an anastomosis between the abdominal portion of the œsophagus and the stomach, and more recently Grey Turner performed the same operation on two patients, with successful results. The operation is difficult, and owing to infection present in any œsophagus with retention it may be dangerous.

Heller made a longitudinal incision through the muscular wall of the abdominal portion of the œsophagus, this procedure being analogous with Rammstedt's operation for congenital pyloric stenosis. Rowlands[26] performed this operation once, with a successful result. Both of these last-

mentioned methods of treatment are dangerous on account of the risk of mediastinitis and peritonitis from leakage at the suture lines, as the tissue of the œsophagus is poorly adapted for suture. Scrimger[27] (1931), believing the condition to be due to spasm of the diaphragm, dilated the œsophageal opening of the diaphragm, pulled the elongated œsophagus down into the abdomen and fixed it there by suturing the edges of the opening in the diaphragm to the thickened œsophageal walls. J. F. Recolde believed that cardiospasm was a disease of muscle tone produced by an inorganic change in the sympathetic system supplying the epicardia. He described an operation in which Auerbach's plexus throughout the entire epicardial region was destroyed, and claimed thereby to effect a radical cure of the condition. The operation consists of decortication of the epicardia from its adventitia and longitudinal muscle in the plane occupied by the plexus. This causes paralytic dilatation of the circular muscle which tonically closes the epicardia and so corrects the obstruction permanently.

The writer strongly advocates the method of dilatation by the hydrostatic dilator of Plummer or by the large metal olive of Moersch. The following case histories illustrate the application and the results of these methods.

CASE I.—J.P.B., a male, aged forty-three years, consulted me on July 4, 1932, on account of loss of weight. At the end of 1928 he had all his teeth extracted, thinking that they were the cause of his loss of weight. His usual weight was 88·2 kilograms (14 stone), but at this time he weighed 63 kilograms (10 stone). After the removal of his teeth he gained weight, presumably because, on account of the loss of his teeth, he adhered to a fluid diet, his weight gradually increasing to 75·6 kilograms (12 stone). He then developed pain in the chest. He could eat a moderate amount of food, but in a matter of ten hours it would be expelled, there being no bitter taste and no acidity. He consulted a herbalist, who gave him some medicine which enabled him to swallow his food, but he still vomited a large amount of mucus three to four hours after his meals. In 1931 he noticed increased difficulty with swallowing; he could not get food down, as it used to stick in his neck, while he noticed that he was worse if he was excited or worried with business difficulties. The trouble gradually got worse; he could neither eat nor drink, his throat "seemed to close up", solids and fluids were equally difficult, and even water used to return with a peculiar bitter taste.

A radiograph of his œsophagus (Figure I) was reported by Dr. Anderson Stuart as follows: "There is a constriction at the lower end of the œsophagus at the cardiac orifice, and there is an enormous dilatation of the œsophagus above this. There was marked delay in the passage of fluids or solids. The X ray evidence indicates achalasia of the cardia."

I dilated the œsophagus with Moersch's dilator, with immediate relief. In reply to a letter (February 4, 1933), he wrote: "My condition is good, everything goes down, and I eat anything that comes along without any restriction whatever. As regards my weight, well, I started off by adding somewhere about three or four pounds weekly immediately after the treatment, and continued to do so until I reached 13 stone 12 pounds, as you know, from under 11 stone."

On February 11, 1936, the patient reported back for reexamination at my request, when radiological examination of his œsophagus showed that there was still some dilatation of the œsophagus and a little delay at the cardia (Figure II). I then wrote to inquire of his present state, and he replied as follows: "I am pleased to advise you that my weight is 13 stone 12 pounds; this I have maintained since your treatment four years ago. In reviewing the general conditions, I find it a pleasure in being able to take anything I eat. I do not diet myself in any way, and the only trouble is, I think I am too fat, as everything I eat agrees with me and shows to good effect, notwithstanding the fact that I do not eat too much."

CASE II.—C.T.F., a female, aged forty years, consulted me on March 1, 1931, complaining of discomfort beneath the lower end of the sternum. She had had dysphagia and had experienced equal difficulty in swallowing fluids and solids. She had lost some weight.

FIGURE I. Case I. Skiagram showing dilatation of the œsophagus extending high up in its thoracic portion.

FIGURE II. Case I. Skiagram showing some delay at cardiac end of œsophagus, but the dilatation of the œsophagus does not extend nearly as high as in the previous figure. Clinically this patient obtained complete relief of symptoms.

On March 2, 1931, her cardiac orifice was dilated with Plummer's hydrostatic dilator; she experienced relief from the sternal discomfort, and her ability to swallow was greatly improved. The history of her condition is, I think, best told in the reports of her radiological examination by Dr. Anderson Stuart. They are as follows:

February 1, 1928: "There is constriction of the lower end of the œsophagus, with dilatation of the œsophagus above. The meal remained in the œsophagus for several minutes. Fluids were delayed as well as solids. The X ray evidence suggests a moderate degree of cardiospasm."

February 25, 1931: "There is still constriction of the cardiac end of the œsophagus, with marked dilatation of the œsophagus above this. There was vigorous peristalsis in the dilated œsophagus. There was a small residue in the œsophagus from a fluid

FIGURE III. Case III. Skiagram showing huge dilatation of the œsophagus as high as the second thoracic vertebra.

barium meal after twenty minutes. From my recollection the dilatation is more marked than at my previous examination, and the delay is certainly greater."

July 17, 1931 (five months after dilatation): "The bolus passes freely down the œsophagus, with only a slight delay at the cardiac orifice. The condition appears to have markedly improved."

November 7, 1933. "The œsophagus is large, but is within normal limits. The opaque bolus passed freely down into the stomach and there is now no evidence of achalasia of the cardia."

CASE III.—A.S.T., a male, aged thirty years, was first seen in consultation on January 25, 1930, complaining of difficulty in swallowing and loss of weight from 65·7 kilograms (10 stone 6 pounds) to 49 kilograms (7 stone 11 pounds) in twelve months. He had also had severe epigastric pain, which had previously been diagnosed as due to gall-stones, and arrangements had been made for their removal. He experienced the

same difficulty in swallowing fluids as he did with solids. He vomited after meals, and the vomitus was bitter. The duration of the symptoms was twelve months, and he felt that his condition was becoming worse. General examination disclosed nothing abnormal except obvious signs of a considerable loss of weight.

Dr. Anderson Stuart, on radiological examination after an opaque bolus, reported a greatly dilated œsophagus, which he considered as being due to cardiospasm. Figures III and IV show reproductions of the skiagrams taken on that date, with huge dilatation of the œsophagus as high as the second thoracic vertebra.

On February 27, 1930, his œsophagus was dilated with a Plummer dilator; he was immediately able to swallow all types of food without difficulty, and on May 30, 1934, he reported himself as being very well and having no difficulty in swallowing.

FIGURE IV. Case III. Another view of the condition illustrated in Figure III.

Since the dilatation his weight had increased to 56·7 kilograms (9 stone). X ray examination after an opaque meal on that date showed the stomach and duodenum to be normal, with an evacuation time of four hours, and no evidence of cardiospasm. After twenty-four hours all the meal had reached the large intestine. On account of the normal emptying of the œsophagus, it was impossible to get a skiagram of it using the ordinary barium meal as the opaque medium. Accordingly, a very thin barium meal was given, and the accompanying skiagram (Figure V) was obtained three months after dilatation. This shows the marked recovery in tone of the œsophageal wall, with a complete absence of dilatation.

CASE IV.—G.H.K., a male, aged twenty-nine years, was under treatment for Hodgkin's disease. On account of difficulty in swallowing, he was referred for radiological examination. Dr. Anderson Stuart's report on this examination of his œsophagus was as follows: "There is a constriction of the distal end of the œsophagus at the

cardiac orifice. There is some dilatation of the whole of the œsophagus above this, and marked delay in the passage of fluids. This appears to be intermittent. The fluid meal was held up for a period of four minutes, the patient experiencing a feeling that the 'fluid would not pass down'. The spasm then gradually relaxed and the meal flowed into the stomach, relief being almost immediate. The X ray evidence definitely suggests a mild degree of cardiospasm of an intermittent nature."

He went to the United States of America for treatment, and was referred to me from the Mayo Clinic for cardiospasm which was associated with Hodgkin's disease, with the following note: "Mr. K. has a definite narrowing at the œsophageal introitus due to scarring from X ray treatment of the glands of the neck. It is therefore

FIGURE V. Case III. Skiagram showing the marked recovery in tone of the œsophageal wall following the use of Plummer's dilator. Note complete absence of œsophageal dilatation.

impossible to pass a hydrostatic dilator for the treatment of cardiospasm. However, he gets complete relief by the passage of a 45 French bougie."

When I first saw him he had complete inability to retain solids; he swallowed them, but vomited them up about fifteen minutes afterwards. A 45 Charrière bougie was passed over a previously swallowed silk thread, and immediately afterwards the patient, unknown to me, partook of a hearty meal of steak and fried potatoes, which caused him no inconvenience and which he did not vomit. He ultimately died of Hodgkin's disease, but up to the time of his death four months later he had no further difficulty with swallowing.

CASE V.—C.R., a male, aged fifty-two years, came for consultation because of inability to swallow food for eight years. His history was as follows. Eight years previously a piece of meat had stuck in his throat and he had coughed it up only after much effort and slapping on the back. About twelve months after this he began to notice difficulty in swallowing solid food. He became unable to eat solid food, as it

seemed to stick at the lower end of the gullet; thus he might get one mouthful down, but then nothing else until he went out and vomited. At first he lost weight rapidly, but his weight was now stationary. He was unable to work, and tired quickly. He had no pain whatever and no other complaints.

The report on radiological examination of his œsophagus was as follows: "There is slight dilatation of the lower end of the œsophagus, with slight delay at the cardio-œsophageal juncture, but no definite cardiospasm. There is no diverticulum present."

On July 9, 1927, œsophagoscopic examination was carried out. The œsophagoscope was passed without obstruction; about five centimetres (two inches) above the cardiac orifice of the stomach there was a dilatation of the œsophagus. On February 13, 1929, attempts were made to pass a stomach tube. At a distance of about 42·5 centimetres (17 inches) from the incisor teeth the further passage of the tube was absolutely and suddenly blocked, so that it would appear probable that the stomach was not entered.

On February 15, 1929, a silk thread was given to the patient to swallow, and twenty-four hours later the Moersch-Plummer bougie was passed over the silk thread without trouble. The patient was made to swallow a large jug of water immediately, which he did without difficulty. He has had no difficulty in swallowing fluids or solids since, and has gained 12·6 kilograms (2 stone) in weight.

CASE VI.—W.C., a male, aged fifty-two years, was seen in consultation with Dr. Susman and Dr. Diethelm on account of difficulty in swallowing, and particularly in swallowing solids. Dr. Anderson Stuart had examined his œsophagus radiologically after the administration of a bismuth bolus, and his reports are as follows:

July 21, 1931: "The œsophagus appears to be very slightly dilated and there was a delay of about eight or ten minutes in the passage of a bolus through the cardiac sphincter into the stomach. Very vigorous peristalsis and anti-peristalsis occurred constantly throughout the whole extent of the thoracic portion of the œsophagus. The radiological evidence suggests a mild degree of achalasia of the cardia."

On January 4, 1932, reexamination of the œsophagus was carried out, the radiological report being as follows: "The œsophagus is now considerably more dilated and the peristalsis is less vigorous. There is still some irregularity of the lower end of the œsophagus. The appearance suggests achalasia of the cardia rather than carcinoma. There was marked delay in the passage of the meal through the cardiac sphincter."

On October 19, 1932, the examination was repeated, with the following report: "There is definite delay in the passage of the bolus and in the passage of cold fluids, and there is dilatation of the œsophagus. Hot fluid appeared to relieve the spasm and the barium then passed freely down into the stomach."

The patient was treated by means of the hydrostatic dilator of Plummer, the dilatation being continued till he complained of pain. He was able to swallow a jug of water immediately after the dilatation, and his swallowing improved for a short period, but his condition then relapsed. He did not consult me again, but went to Europe for treatment. He consulted Professor Lothiesen, who passed his instrument and gave the patient immediate relief. The patient was so grateful that he brought back the Lothiesen dilator with him, and through his courtesy and that of Dr. R. A. Money, I was able to inspect it. It is a beautifully made instrument, somewhat on the principle of a Kollmann's urethral dilator. It has the distinct disadvantage that it is passed blindly into the cardia, and unless it is passed under fluoroscopic control it is impossible, with an elongated œsophagus, to be sure when the dilating blades are in the cardia. It could be easily adapted, by fitting a tubular spiral guide, to render it capable of passage over a previously swallowed silk thread. It would be then similar in principle to the Moersch-Plummer bougies.

CASE VII.—E.C., a female, aged sixty-four years, came for consultation on April 11, 1935. She complained of inability to swallow solid foods for seven years and loss of 18·9 kilograms (3 stone) in weight. There had been no previous illness and the onset of the trouble was said to be caused by worry. She had noticed difficulty in swallowing her food and could feel any solid food stick behind the lower end of her sternum. Shortly afterwards a sensation of obstruction developed, while her food would regurgitate. She experienced no pain whatsoever, and found that she could swallow fluids by taking mouthfuls at a time and waiting till the sensation of blockage disappeared.

Throughout the past seven years the patient had been under treatment, taking various medicines for relief of her condition, but without success. She had been told that her symptoms were due to reflex nervous causes. Nutrition for the past seven

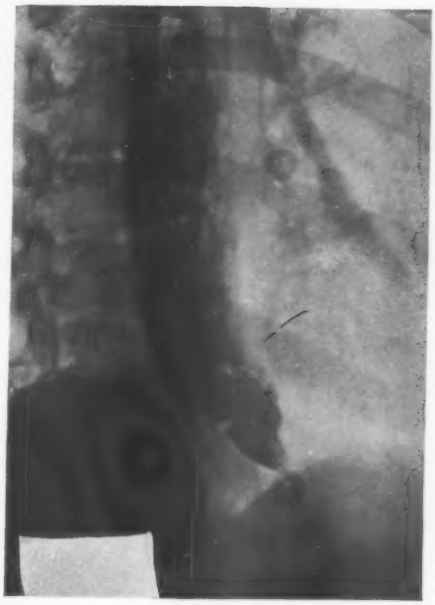

FIGURE VI. Case VIII. Skiagram showing characteristic appearance of cardiospasm, before dilatation.

years had been maintained almost solely by coffee made with milk, and beef tea. At various times she had tried to take semi-solid foods, but these were always regurgitated. At no time had the patient had any hæmatemesis or melena, nor any alteration in her voice. She had had an irritating cough occasionally when the food would become arrested.

On April 8, 1935, radiological examination following the administration of an opaque bolus revealed obstruction at the cardiac end of the œsophagus. The œsophagus was evenly dilated and gradually narrowed to a point. A couple of small shot tied to a piece of silk were given to the patient to swallow. The silk on a reel was anchored to her gown.

On April 10, 1935, the shot passed the cardiac end of the œsophagus, the silk was cut and the œsophageal dilator passed into the stomach. The patient was returned to the ward in good condition. Later on she was able to swallow baked custard, taking small spoonfuls at a time.

Reexamination on April 11, 1935, showed: "Passage of bolus is still blocked, but the patient is able to swallow jelly and custard without any uncomfortable sensations. The radiographic appearances are much changed since the previous examination."

The patient was seen again on June 14, 1937, when her general condition was better and her weight was 53 kilograms (8 stone 7 pounds). She still complained of some difficulty in swallowing, but when she was asked to have another X ray examination of her œsophagus or another dilatation she said that she was quite comfortable and refused further treatment.

CASE VIII.—S.M.V., a female, aged forty-three years, consulted me because of dysphagia and loss in weight of 12·6 kilograms (2 stone). In May, 1932, she had noticed that if she ate quickly she had difficulty in swallowing and had to stop. During the remainder of 1932 and 1933 she became gradually worse, and at the beginning of 1934 she consulted her medical adviser because of difficulty in swallowing meat and bread. She noticed increasing difficulty with solids during the next twelve months, and finally could take only fluids. She repeatedly vomited a bitter material mixed with much mucus.

I saw her on September 10, 1936, but because of language difficulties her history was difficult to obtain. A radiograph of her œsophagus after an opaque meal showed the characteristic picture of cardiospasm (Figure VI). Her cardia was dilated with a Moersch dilator, and she immediately swallowed a jug of water. I saw her a month later, and she then had no difficulty with meats or

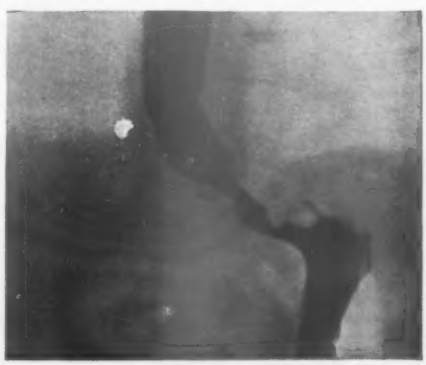

FIGURE VII. Case VIII. Skiagram of œsophagus after dilatation.

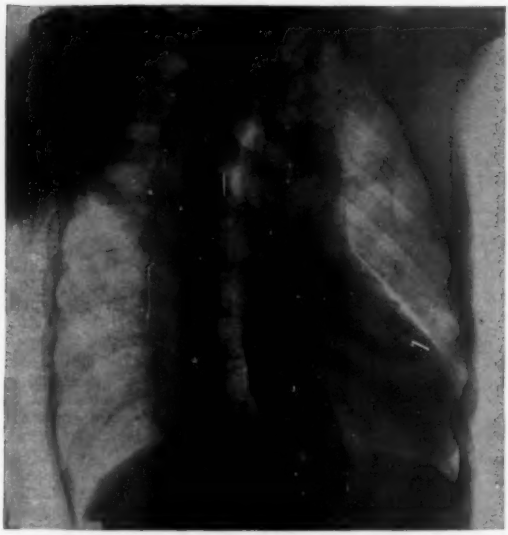

FIGURE VIII. Case IX. Skiagram of the œsophagus, suggesting a diagnosis of cardiospasm.

bread, and had gained 4 kilograms (9 pounds) in weight. I saw her again on December 4, 1936, when she had gained a further 4·9 kilograms (11 pounds), and was having no trouble at all with swallowing. Figure VII shows a skiagram of the œsophagus after dilatation.

The following case, though not one of cardiospasm, is nevertheless of interest in this connexion, as cardiospasm was difficult to exclude both on clinical grounds and on the radiographic evidence.

CASE IX.—D.S., a male, aged seventy years, noticed four months previous to consultation that he could not take his usual amount of food and could not eat any meat. He first noticed difficulty in swallowing solids, then later had equal difficulty with fluids, while he had lost 9·4 kilograms (one and a half stone) in weight in the previous six months. Clinical examination confirmed the patient's statement that he had lost much weight. No mass was palpable in the abdomen, but an analysis of the history suggested the presence of malignant disease. He was referred for radiological examination, and the following was the report received: "There is a very marked obstruction at the cardio-œsophageal orifice, with great dilatation of the œsophagus above. A considerable amount of the meal remains in the œsophagus two hours after its ingestion, and there is still some present at the end of four hours. Insufficient of the meal passes into the stomach to render the examination of this satisfactory. The diagnosis lies between cardiospasm and malignant disease of the upper end of the stomach close to the cardio-œsophageal orifice. It is impossible to be certain radiographically. The amount of delay suggests cardiospasm, though there appears to be some irregularity just below the cardio-œsophageal orifice, as indicated by the arrows. An œsophageal examination might settle this question" (see Figure VIII).

I felt that dilatation by Plummer's sounds over a swallowed thread might be pre-

FIGURE IX. Case IX. Skiagram showing advanced carcinoma of the cardiac end of the stomach.

ferable. This was done, and after a second radiographic examination the radiologist reported: "There is still considerable delay at the cardio-œsophageal orifice. The meal, however, passes down and shows an extensive, constant, irregular narrowing of the cardiac end of the stomach, the appearance of which is typical of an advanced carcinoma in this region" (Figure IX). The patient obtained so much relief from the dilatation that he refused anything in the way of a jejunostomy, and came for a further dilatation on three occasions at intervals of about a month before he died.

SUMMARY.

1. Eight cases of cardiospasm, five in males and three in females, are reported, with the subsequent history of each.

2. Dilatation in each case was carried out on one occasion only.

3. The method of dilatation over a previously swallowed silk thread, with either a Plummer hydrostatic dilator or a Moersch-Plummer bougie, is advocated.

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PROSTATIC SURGERY: THE QUESTIONS OF HÆMOSTASIS AND SECONDARY HÆMORRHAGE.[1]

By Sir Henry Newland,
Adelaide.

Hæmorrhage calling for treatment after prostatectomy usually occurs within forty-eight hours of the operation, and is most often arterial, the spouting points being situated on the posterior rim of the crater left by the removal of the gland. When venous, it is commonly due to the enucleating finger having entered the wrong plane and having injured the prostatic plexus. It is rare for post-operative hæmorrhage to occur from the suprapubic wound in the bladder, but in one case I was compelled to expose the bladder again and ligate a spouting vessel in its wall.

Post-operative hæmorrhage has engaged the attention of surgeons ever since Freyer popularized prostatectomy. Hæmorrhage apparently at no time caused that surgeon any concern. Irrigation with a hot solution of potassium permanganate was the only measure he employed to arrest whatever bleeding there might be after the enucleation. Further, if reference be made to the papers on prostatectomy which he was wont to publish from time to time, it will be found that the mortality lists contain no case in which death is attributed to hæmorrhage. In a series of 1,550 cases the death rate was a little more than 5%; in the last 200 cases it was reduced to 3%. It is quite true that the deaths directly due to hæmorrhage are few. Thompson-Walker could only find two such deaths in the many patients who had undergone prostatectomy at Saint Peter's Hospital. In 39 private patients of my own there was one such death. It is not open to doubt, however, that hæmorrhage is frequently an important factor in deaths ascribed to shock, collapse, asthenia, renal failure, bronchitis, bowel complications and other sequelæ. In my small series two deaths occurred from pulmonary embolism, one on the nineteenth and the other on the twenty-eighth day. Hæmorrhage may in these two cases have been a factor in the formation of the thrombotic source of the embolus. In another case the post-operative bleeding was arrested by exposing the prostatic rim and ligating the bleeding point.

Freyer's figures indeed give no idea of the anxieties which must frequently have beset the house surgeons and nurses who looked after his patients. While hæmorrhage and its effects after operations on other organs are usually obvious enough, after prostatectomy the rôle of hæmorrhage in producing untoward symptoms is not seldom obscured owing to the observer

[1] Delivered on the occasion of the symposium on prostatic obstruction, held during the eleventh annual meeting of the Royal Australasian College of Surgeons, Sydney, March, 1938.

finding comfort in the false belief that what is flowing from the bladder is not blood with a little urine, but much urine with a modicum of blood.

In the operation of suprapubic prostatectomy as performed for over twenty years, two principles which govern the removal of a diseased organ were not applied. The arterial supply of the prostate was not controlled prior to, or in the course of, its removal, nor was any attempt made to restore the continuity of the canal which traversed the gland. Thompson-Walker in 1921 applied the first principle when he employed larger abdominal and vesical incisions, which enabled him to identify and ligate bleeding points on the edge of the prostatic crater. However, even this procedure is not proof against recurrence of hæmorrhage. Some years later Harris applied both principles in a technique in which sutures were employed to stem the hæmorrhage from the prostatic crater, and at the same time to reconstruct the urethra. Harris's operation has revolutionized the performance of prostatectomy, but, nevertheless, the bugbear of hæmorrhage still exists. In the most capable hands sufficient bleeding sometimes occurs to block the urethral catheter, fill the bladder with clot, and compel reopening of the suprapubic incision.

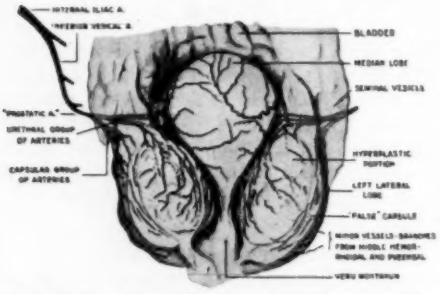

FIGURE I. After Flocks. Semi-diagrammatic illustration of distribution of urethral and capsular groups of arteries within prostate gland. Note course of urethral arteries—they enter at prostatic-vesical junction. Note that urethral arteries supply major portion of hypertrophied lobes.

It will be noted that in the Thompson-Walker and Harris operations no attempt is made, as in thyreoidectomy and other visceral extirpations, to control the blood supply by ligation at an early stage in the procedure. A variable amount of bleeding must necessarily occur after enucleation of the prostate before the means to check it are employed. Is it possible by any means at our disposal to improve the Harris operation, and minimize the loss of blood in the course of and subsequent to prostatectomy? Is it possible to secure by ligature the arterial supply of the prostate before proceeding with the enucleation? I do not know, but a paper by R. H. Flocks[1] throws some light on the question.

The prostate receives its main blood supply from the inferior vesical artery. Small branches to the inferior part of the gland are derived from the middle hæmorrhoidal and the internal pudic. There is no single prostatic artery, but there are about four or five branches arising from the inferior vesical artery, which course more or less parallel to each other, and are for convenience grouped together descriptively and called the "prostatic artery" (see Figure I). The prostatic artery gives rise to two groups of vessels which distribute themselves in a fairly regular manner throughout the prostate. These are best named the urethral and the capsular group. The urethral or penetrating group immediately penetrates the tissue at the prostatico-vesical junction. Here it sends some branches into the lateral lobe, and then distributes itself along the bladder neck and the urethral surface of the prostate to supply the vesical neck and inner portion of the prostate. The other group, the so-called capsular or non-penetrating group, courses along the postero-lateral surface of the gland, and sends branches ventrally and dorsally to supply the outer portion of the prostate.

With the advent of age and the often concurrent hypertrophy of the prostate, changes in the anatomy of the gland are associated with changes in the arrangement and relative importance of the urethral and capsular arteries. The vast majority of benign enlargements of the prostate are central, next to the urethra, and it has been found that the hypertrophied portions are supplied in the main by the urethral group of arteries. The median lobe is supplied almost in its entirety by urethral arteries coming in on each side at the prostatico-vesical junction at the level of the clefts between the lateral and median lobes. Practically the entire hypertrophied portion of the lateral lobe is also supplied by the urethral group of arteries and its branches. Only the extreme outer portion of it is supplied by branches from the capsular group. In very large prostatic hypertrophies the capsular branches are larger and more numerous, and there is more abundant anastomosis between these and the branches from the urethral group.

Reference to the illustration shows that it might be possible to pass a suture at an appropriate distance on each side of the middle lobe, and thus control most of the arterial hæmorrhage which ordinarily occurs during and after enucleation. Experience will show whether this course is possible. If so, it will be a further step in perfecting the technique of prostatectomy.

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THE CAUSE AND DIAGNOSIS OF PROSTATIC OBSTRUCTION.[1]

By Richard G. S. Harris,
Sydney.

Prostatic obstruction may be defined as a mechanical or physiological hindrance to the free passage of urine, occurring in the region of the bladder neck and prostatic urethra. It is commonly found in men from fifty years of age onwards, though cases in the fourth decade are by no means rare.

The obstruction may be induced by inflammation of the prostate gland, or, more commonly, by the association of an adenomatous condition of the prostate, with hypertrophy and adenomata of the urethral and suburethral glands. It is generally agreed that the term "hypertrophied prostate" is a misnomer, and that the pathology lies in the formation of adenomata within the gland, whilst the surrounding prostatic tissue is compressed into the surgical capsule. True hypertrophy of the prostate is, however, occasionally found.

From animal experiments by Lower and his associates, and from their more recent clinical application, there appears to be considerable evidence in support of the theory, that endocrine imbalance plays an important part in the causation of prostatic enlargement. It would appear that the relationship between the pituitary and testicular hormones is altered, and that the effect of the former on the prostate becomes unrestrained. There is also, however, much clinical evidence to show that infection, congestion and neoplasm, as well as endocrine imbalance, play their parts, and it is impossible to place any one of these factors in a position of prime importance.

Reverting to the original conception of obstruction, the mechanical element can be demonstrated by an inspection of the prostatic urethra and bladder neck. Encroachment on, and partial obliteration of, the urethral lumen, with an associated lengthening and tortuosity of the prostatic urethra, are seen in those cases in which the enlargement is mainly intraurethral. Varying degrees of middle lobe enlargement are generally present, either alone or with the lateral lobe enlargement, and may form an effective ball-valve where the enlargement is mainly subtrigonal and intravesical.

By the physiological impediment, I imply the inability of the trigonal muscle readily to pull open the internal sphincter. This occurs especially in chronic inflammatory lesions, the so-called "median bar", and in those types of obstruction which are mainly subtrigonal. I should particularly like to draw attention to the marked hypertrophy of the interureteric ridge, which is found associated with these conditions. This must be interpreted as evidence of hyperactivity of trigonal muscle, and it may advance to such a degree

[1] Delivered at the symposium on prostatic obstruction on the occasion of the eleventh annual meeting of the Royal Australasian College of Surgeons, held in Sydney, March, 1938.

that, in itself, it becomes an added mechanical obstructive factor. Harry Harris was of the opinion that hypertrophy of the interureteric ridge was a corollary of mid-line obstruction, and that many of the patients would not obtain relief of symptoms unless surgical attention were directed to the secondary, as well as to the primary, cause of the obstruction.

In passing, I believe that when gross hypertrophy of the interureteric ridge is present, or when a *bas fond* is found in association, transurethral resection is contraindicated, as the removal of tissue from the prostatic urethra is insufficient for cure, whilst the dangers of intravesical resection are too great to be countenanced.

The actual size of the prostate appears to bear but small relationship to the degree of obstruction. The encroachment on, and the lengthening and tortuosity of, the prostatic urethra, the presence or absence of a ball-valve, and the impairment of the function of the trigonal muscle are of paramount importance.

For the sake of brevity, I propose to mention only the prostate, when, in the strict sense, the urethral, the suburethral or the subtrigonal glands should also be included.

The prostate gland may become the site of innumerable pathological processes, each and all of which may cause obstruction in varying degrees. Many of these conditions are amenable to purely medical treatment, and I shall accord them but passing mention. I refer particularly to acute infections secondary to posterior urethritis, to acute hæmatogenous infections which do not proceed to pus formation, to those obscure cases of phlegmonous periprostatitis, and to certain cases of œdema of the prostate. Administration of heat and urinary antiseptics, the judicious use of rectal massage, and the temporary use of an inlying catheter will cure the large majority of these cases.

The conditions which call for surgical interference fall into the simple classification of inflammatory and non-inflammatory, and the latter may again be divided into malignant and non-malignant.

Prostatic abscess, whilst frequently of Neisserian origin, occurs not uncommonly as a hæmatogenous infection following on acute systemic disease. From the absence of any previous history, from the rapid onset of urinary difficulty and strangury, perineal pain and pyrexia, and from the findings on rectal examination, the diagnosis is usually obvious. Incision and drainage by the perineal route afford the most satisfactory results and have never, in my experience, been followed by fistula formation.

Certain chronic inflammatory conditions of the prostate are encountered in which the diagnosis may be impossible apart from cystourethroscopic inspection, accompanied by palpation of the bladder neck *per rectum*. The main symptoms are frequency of micturition, with or without difficulty, especially in the commencement of the act, and some diminution in the size and force of the stream. Residual urine up to 120 cubic centimetres (four fluid ounces) or more may be present. Renal colic, the so-called "reflux" renal colic, may rarely be the only prominent early symptom, when intravenous urography gives a typical picture of generalized dilatation of the upper urinary tract. Confirmation by cystoscopic examination is easily obtained. A percentage of these cases will respond admirably to rectal massage, and in these the obstruction will invariably be found to be of a soft and œdematous

nature. Where the obstructing bar is firm, however, and there are no intravesical complications, the treatment of choice would appear to be transurethral resection.

Another somewhat analogous condition is that in which the internal urethral orifice is tightly contracted and there is a generalized sclerosis of the internal sphincter. The operation described by the late Harry Harris under the title of "Posterior Segmental Block-Excision of the Bladder Neck" offers, perhaps, the greatest prospect of complete cure. These two conditions have been described by French writers as *prostatisme sans prostate.*

In general, I do not believe in the indiscriminate cystoscopic examination of patients suffering from prostatic obstruction. As a rule, the diagnosis is obvious from the symptomatology, and from rectal examination. When rectal examination is inconclusive, or when the examination suggests only a moderate degree of enlargement, the pathological nature of which is in doubt, cystoscopic examination then affords the only means of arriving at a satisfactory diagnosis, and of evaluating the most appropriate method of treatment, whether suprapubic prostatectomy or transurethral resection. Should calculi be present, their removal may be accomplished at the time of the major procedure, and I cannot too strongly condemn the practice of removing vesical calculi in men of prostatic age without eliminating the common predisposing cause of their formation, namely, lower urinary obstruction. Where a moderate to a great degree of adenomatous enlargement is present, I believe that the Harris method of prostatectomy is the ideal treatment.

I should like to turn now to the consideration of malignant disease of the prostate, and here, I am afraid, we have to confess a degree of failure in our methods. The condition appears to be hopeless of cure in the present state of our knowledge, at whatever stage the patient may present himself for treatment. We appear to be just as helpless when the condition is found in its early stages as when a hard indurated mass is discovered *per rectum.* Of the causation, as with cancer in other parts of the body, we are equally ignorant. It is suggested that chronic inflammation may be a predisposing cause. It is an undoubted fact that malignancy may occur within an adenomatous enlargement, in which case, however, a clinical cure may generally be effected by enucleation of the prostate. This type of malignancy does not usually tend either to local recurrence or to the formation of metastases, and can generally be diagnosed only by microscopic section of the removed prostatic tissue.

As a rule, the diagnosis of advanced carcinoma of the prostate is a simple matter on rectal palpation. The hard craggy nodules, irregular in their distribution, the extension of the mass towards the lateral walls of the pelvis, to which it becomes attached, the accompanying rigidity of the bladder base and, lastly, the extension downwards along the urethra, are quite typical. In early cases, it may be a difficult matter to differentiate between a small fibrotic and a small, hard, malignant prostate. Occasionally prostatic calculi may be a source of error in diagnosis, but the presence of an extension of the growth along the urethra is a significant sign, suggestive of malignancy. When a small, hard nodule, not infrequently solitary, is felt on the posterior surface of the prostate, it may be impossible without cystoscopic

and radiological examinations and palpation *per rectum* against the cystoscope, to arrive at an accurate diagnosis.

In general, in the early stages of malignancy, the final diagnosis rests on a combination of rectal, cystoscopic and radiographic examinations, and an added indefinable factor, experience. The most satisfactory treatment, whatever the diagnosis, in all these small, hard prostates is the same—transurethral resection. Examination of the tissue so removed will set aside any doubt as to the nature of the condition. A fact that would appear to be of some assistance in prognosis is that the degree of malignancy tends to vary in inverse ratio to the size of the prostate.

The symptoms of malignant disease of the prostate show little difference from those associated with the non-malignant condition. There is, perhaps, a rather shorter interval from the onset of symptoms until the development of pronounced distress.

My practice is to advise against any surgical interference until pronounced symptoms are in evidence, and then to decide upon the most suitable type of operation after cystoscopic inspection.

To summarize the symptomatology of prostatism in general, the sequence is typical and definite. The age of onset may be as early as the fourth decade, though it is unusual for patients to present themselves for treatment at this age. Some hesitancy in the commencement of micturition and some delay and dribbling in the completion of the act, associated with a diminution in the size and the force of the stream, are usually the earliest presenting signs. Spasmodic attacks of frequency, both by day and by night, occur, and are gradually replaced by a permanent and increasing nocturia, which may progress to a virtual incontinence. Urgency and a constant desire to pass urine, with an ever-present feeling of incompleteness of the act and suprapubic discomfort, are the usual sequence. An attack of urinary retention is often precipitated by the inaccessibility of a suitable place for urination, or by the consumption of alcohol, especially beer, even in moderate quantities. Attacks of retention may be followed by periods of comparative ease, but the severity of the symptoms gradually increases, whilst the intervals of relief are shorter. Urgent and precipitant micturition, with straining and hæmaturia, are frequently associated with attacks of congestion of the prostate and infection of the residual urine, which has now become permanently established. Complete retention is the natural sequel.

Moderate hæmaturia is not a sign to which any particular significance can be attached, and does not appear to be associated any more frequently with a malignant than with a non-malignant condition. Gross hæmaturia, however, even in the presence of the other signs of prostatism, should always demand cystoscopic investigation before operation is undertaken, in order to eliminate the possibility of intravesical tumour.

CELLULITIS OF THE COLON: A MANIFESTATION OF DIVERTICULITIS.

By W. A. Hailes,

Melbourne.

In the classification of the complications of diverticulitis reference is consistently made to swelling of the bowel wall and obstruction of its lumen, but the same prominence is not directed to this pathological condition as is given to the inflammatory reactions inside the peritoneal cavity. Obstruction from inflammatory reaction and swelling of a length of colon, occurring at the time of the acute inflammation, is the only type of intestinal obstruction that I have encountered in diverticulitis. Chronic obstruction from cicatrization, bands or adhesions, occurring late in the disease, has not been encountered as a cause of intestinal obstruction in recent years in the Pathological Department of the Royal Melbourne Hospital. I refer here to true obstruction, and not to impaired function of the sigmoid colon.

Pathology.

The intraperitoneal complications of diverticulitis are well recognized and are more numerous than any of the other complications; nevertheless, I think all will agree that even in those cases with pelvic inflammation and suppuration the greatly thickened sigmoid colon is just as striking as the inflammation in the peritoneum. This swelling is much more extensive than that usually seen in the caecum in association with acute appendicitis in those cases in which the caecal wall is involved, a statement which applies both to the induration of the bowel wall and to the length of bowel involved. The acutely inflamed sigmoid colon appears quite often to be the origin of the inflammatory exudate, and not the diverticula. There is a large area of visceral peritoneum inflamed.

Because of the association of the swelling of the bowel with acute inflammation in one or more diverticula in these cases, we can assume that the inflamed diverticulum is responsible for the cellulitis, and this cellulitis may be the outstanding lesion present. It is, indeed, often impossible to localize one diverticulum that has perforated, or is more acutely inflamed than many of its fellows. The cellulitis may be localized to one portion of the sigmoid colon, or involve the whole sigmoid, or even in extreme cases the whole of the descending colon; the degree of swelling of the bowel may be extreme and it is not at all uncommon to see a sigmoid loop so swollen and indurated that the loop in question is unmanageable surgically if obstruction has supervened.

Let us now consider the end-result of the cellulitis. Cellulitis of the bowel pursues the same course as cellulitis elsewhere in the body, and may

lead to (*a*) resolution, (*b*) local abscess formation, with or without rupture, or (*c*) obstruction.

The subject of resolution will be summarily dismissed. It is apparent that it must occur in all those cases of diverticulitis with pelvic suppuration in which recovery takes place after operation.

Local abscess formation, that is, localization with, in most cases, rupture into the bowel lumen, occurs quite often, in many cases with discharge of pus. In Case I (to be described below in detail) the pus was discharged *per rectum,* while in Case III I excised a loop of sigmoid colon in error, mistaking a localized intramural abscess for a carcinoma.

The incidence of obstruction in diverticulitis is not high, but such cases as occur are regarded as difficult to manage, chiefly because of the extent of the bowel involved. The induration of the bowel wall due to the inflammatory reaction, leading as it does to shortening and rigidity of the mesentery, renders mobilization difficult.

It has been said that diverticulitis does not give rise to obstruction, but this is not so. Obstruction does occur and is partly of mechanical origin, due to the swelling obliterating the lumen over a long segment of bowel, and partly paralytic, because it is not reasonable for this long segment to undergo efficient peristalsis if all its coats are inflamed.

The diagnosis of diverticulitis has always to be made from: (*a*) other inflammatory lesions in the lower abdomen, and (*b*) other causes of obstruction in the lower bowel.

So far as inflammatory lesions are concerned, the history of an acute onset, the local and general signs of inflammation and the local pain and tenderness with leucocytosis and tenderness on rectal examination, will help to indicate the probable diagnosis of diverticulitis, while their degree of severity will indicate whether expectant or operative treatment is required.

Most surgeons will recall many cases where no gangrenous or perforated diverticulum was present, and in which operation probably fulfilled no useful purpose. Nevertheless, if the inflammatory signs are sufficiently acute, laparotomy is essential in these cases, for nobody can, without laparotomy, differentiate between those cases in which the cellulitis is the outstanding factor and those with perforation and peritonitis. Moreover, as diverticulitis is a disease of the later decades, if the patients with gangrene or perforation are not treated early by exploration and drainage, the mortality is very high. In view of this policy which necessarily has to be pursued, some patients will be submitted to laparotomy who would have recovered without operation.

Diagnosis from other causes of lower bowel obstruction practically means the exclusion of carcinoma. The following are the principal criteria that point to a diagnosis of diverticulitis:

(*a*) The short history and the local and general signs of inflammation, that is, local tenderness and rigidity, and elevation of the temperature and pulse rate. The general signs may be mild in degree. Findings such as a temperature of 35·6° C. (99·6° F.) and a pulse rate of 96 may easily occur.

(*b*) The occurrence of difficulty and pain in the course of attempts to administer enemata to a patient without gross abdominal distension is perhaps significant.

(*c*) The result of the enema reveals the degree of obstruction. In many of these cases of cellulitis of the colon, if the length of bowel involved is not extreme, the bowel can be kept cleared by means of enemata because the obstruction is incomplete.

(*d*) The significant findings on radiological investigation are as follows: (i) the presence of diverticula; (ii) the length of the filling defect—a very long filling defect with a short clinical history is suggestive; (iii) the degree of obstruction as indicated by dilatation of the colon proximal to the filling defect.

The presence of diverticula merely indicates the possibility of diverticulitis. A long filling defect, associated with a short history, inflammatory

FIGURE I. Showing a long filling defect associated with diverticula. In this case carcinoma was present in an area of diverticulosis. A long filling defect does not indicate an inflammatory lesion; it only hints that such is possible.

signs and the presence of diverticula, is significant, but affords no more than a ray of hope that the condition may not be malignant. A short history, a long filling defect, diverticula, and no evidence of dilated bowel proximal to the filling defect, in a case with local pain and tenderness and inflammatory signs, will also suggest an inflammatory lesion, and, in the absence of obstruction, indicate delay and expectant treatment, with control by periodic radiological observation.

The specimen and skiagrams of one patient show that the diagnosis from carcinoma may be very difficult. This patient had a short history, local pain and tenderness, no general inflammatory signs, and no palpable mass. Radiographic examination showed a long filling defect in the sigmoid loop

(see Figure I); the loop was studded with diverticula. The clinical diagnosis was carcinoma; the radiographic, diverticulitis.

The mass removed at operation was a colloid carcinoma, with superficial ulceration and flat infiltration of a segment of the bowel at least 3·75 centimetres (one and a half inches) long. There was a commencing spread on the peritoneal surface.

TREATMENT.

A good many patients with cellulitis of the colon without obstruction are doomed to laparotomy because of the risk of perforation of a diverticulum; in the absence of the latter, opinion may possibly differ as to whether drainage is necessary.

In the presence of purulent fluid I would always employ drainage, regardless of whether a gangrenous or perforated diverticulum were detected.

Some patients treated expectantly will discharge pus *per rectum* with resolution. It is in these cases with a progressive obstruction or rapidly developing obstruction that most difficulty is encountered. If the degree of obstruction threatens life, colostomy will be necessary. It is here difficult, indeed often impossible, because of the cellulitis of the sigmoid colon, to perform a left iliac colostomy, and it is also inadvisable because subsequent mobilization of the descending colon will be embarrassed. It is much better to perform a transverse colostomy and subsequently check the pathological condition and the patency of the lumen of the colon by radiographic examination.

It is my firm conviction that if the case is one of cellulitis of the colon, secondary to diverticulitis, the disease in the colon will in most cases, with or without colostomy, become localized and discharge into the lumen of the bowel, and that the swelling will subside like a cellulitis anywhere else. There is such a relatively wide lumen in comparison to the thickness of the bowel wall that appreciable stenosis does not occur and chronic obstruction does not result.

Extensive bowel resections are seldom, if ever, necessary. After a reasonable interval of time a clysma will reveal the bowel lumen patent. The colostomy can then be closed, if it has been necessary to perform one for obstruction, and the bowel can be allowed to conduct and function normally. As regards the region of the colon likely to be involved, all the cases which have come under my notice, except one, have been in the sigmoid. In Case V the lesion was in the middle of the transverse colon, and although it was thought to be carcinomatous at the time, subsequent events render this diagnosis most improbable.

The difficulty that may occur at operation of distinguishing carcinoma with obstruction from diverticulitis is well known, but this condition is seldom now seen at operation, because the risk of exploration in obstructive cases is so great that a blind colostomy is performed.

My experience, from days when I did carry out exploration in these cases more often than I do now, is that the more extensive the cellulitic reaction, the more likely is the condition to be inflammatory and not malignant.

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A series of six cases will now be described in detail.

CASE I.—The patient, a male, aged forty-nine years, was first seen on April 10, 1925. For the previous seven days he had had abdominal pain, colicky in nature, with difficulty in obtaining an efficient bowel action, although for a while he had passed some flatus. Enemata yielded unsatisfactory results, and the condition culminated in gross abdominal distension, vomiting and hiccup and complete intestinal obstruction, which when the patient was first seen had been present for two and a half days.

There had been a previous operation for acute appendicitis with peritonitis some years previously, and two scars, one in the right iliac fossa and the other in the mid-line below the umbilicus, were now the site of ventral herniæ.

On examination the temperature was 37·5° C. (99·4° F.) and the pulse rate 132. The patient was very ill indeed. He was a stout, florid male with troublesome hiccup. The abdomen was grossly distended, with both flanks bulging, suggesting large bowel obstruction. On rectal examination nothing abnormal was detected.

Operation revealed a large tumour in the sigmoid loop so inflamed and fixed that the sigmoid and descending colon were almost unmanageable surgically. With difficulty a tube was sutured into the commencement of the sigmoid colon and the abdomen closed. A diagnosis of obstruction from a malignant growth of the sigmoid colon was considered established.

Despite the facts that the tube drained well and that all the distension disappeared, the temperature mounted to 38·3° C. (101° F.); but the patient's general condition was satisfactory. It was not till there was a copious discharge of pus *per rectum* twelve days after operation that the temperature and pulse rate fell to normal. Some weeks later the sigmoid colon was resected by another surgeon, who kindly showed me the specimen. Although the wall was still rigid, the lumen was patent, the mucosa was intact and diverticula were present.

This was clearly a case of obstruction of the colon due to cellulitis of the sigmoid colon. Resolution followed abscess formation and discharge of pus *per rectum*.

CASE II.—The patient, Mrs. A., aged sixty years, was first seen on October 10, 1927, complaining of pelvic pain and discomfort for fourteen days, the condition having originated, according to the patient, during a long motor ride in cold weather. At first the condition was associated with pain when the bowels acted; later it was followed by great difficulty in getting the bowels to act at all, and was associated with abdominal distension. The temperature for ten days had varied between 37·8° C. (100° F.) and 38·3° C. (101° F.) each night. The pulse rate was 96.

On examination, the abdomen was not now distended, and there was tenderness above the *symphysis pubis* and in the left iliac fossa. On vaginal examination there was a tender mass on the left side, which was thought to be an old quiescent pyosalpinx recently activated. On rectal examination the same tender mass was felt, and was thought to be outside the bowel wall.

At laparotomy the mass was seen to be in the sigmoid colon, which was with difficulty delivered; it was considered to be a carcinoma and a resection with end-to-end anastomosis was performed. Convalescence was uneventful.

On examination, the specimen of sigmoid colon was studded with diverticula, and the mass was an intramural abscess which certainly would have discharged *per rectum* had operation been delayed. Operative treatment would then have been unnecessary.

The pathological sequence here would seem to have been: diverticulitis; cellulitis of the bowel; resolution with abscess formation.

CASE III.—The patient, A.W.McC., female, aged forty-three years, was first seen on June 9, 1927. She had experienced sudden onset of colicky lower abdominal pain, associated with vomiting and tenesmus and the passage of a little blood and mucus. The patient had retired at night perfectly well and had awakened in the early morning with these symptoms. An efficient hospital sister could not give the patient an enema, as the fluid would not run in.

On examination, the temperature was 37·5° C. (99·4° F.) and the pulse rate 84. The patient was stout. Her abdomen was a little distended, but soft, with tenderness in the hypogastrium and the left iliac fossa. No mass was palpable, but the abdomen was very obese. On rectal and vaginal examination no mass was palpable, but there was tenderness on pressure in the left side of the pelvis and the pouch of Douglas.

A diagnosis of obstruction in the lower portion of the sigmoid colon was made.

Operation revealed the most extensive degree of cellulitis of the colon that I have encountered. The whole of the sigmoid and descending colon up to the splenic flexure was inflamed and fixed and so swollen that it was impossible to do anything with it, and to drain that segment would have been futile.

A mid-transverse colostomy was performed, the patient's condition was soon satisfactory, and on August 15, 1927, Dr. Hewlett reported that radiological investigation revealed no obstruction to the passage of the opaque medium, but that the sigmoid and descending colon did not distend well. The clysma revealed a diverticulosis of the sigmoid colon. The colostomy was allowed to close, and now, ten years later, the patient has no disability, the bowels acting normally with the aid of a little paraffin.

Associated with a diverticulosis this patient had a most extensive cellulitis of the colon which resolved following colostomy. No discharge of pus was noted in this case, though it may have occurred.

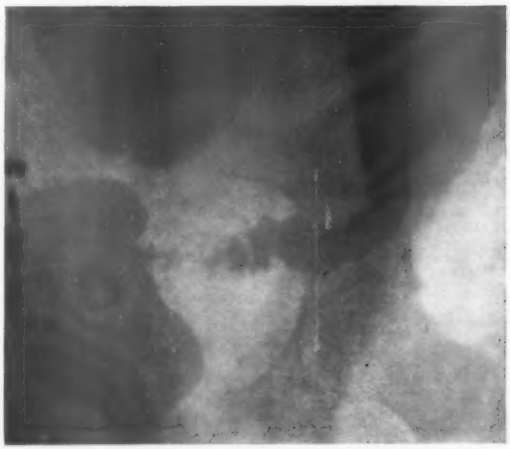

FIGURE II. Case IV. Showing the length of the filling defect and the presence of diverticula.

CASE IV.—The patient, J.R.B., male, aged sixty years, was first seen on March 29, 1935. His complaint was one of abdominal pain for a few days, relieved a little by taking aperients; for the past twenty-four hours there had been no bowel action, and colicky pain had been severe till a little flatus had been passed an hour before examination. During the past twenty-four hours he had several times passed a little blood and mucus *per rectum*.

On examination, the temperature was 37·2° C. (99° F.) and the pulse rate 92. The patient was rather florid and stout, his abdomen was a little distended, both flanks were full and resonant, and the whole abdomen was rather tympanitic. There were tenderness and a vague mass in the left iliac fossa. On rectal examination no abnormality was detected.

The case was considered to be one of carcinoma of the colon, and this report was confirmed by radiographic examination (see Figure II).

It is important to notice, however, the long extent of the filling defect, a point worthy of some consideration. This patient passed into the care of another surgeon, a transverse colostomy was performed, and before anything more could be attempted a coronary thrombosis occurred. When the patient was convalescent from this, further

FIGURE III. Case IV. Showing the lumen of the bowel restored after some months during which the patient was convalescent from a coronary thrombosis.

FIGURE IV. Case V. Showing the large length of the filling defect.

FIGURE V. Case VI. Showing the site of the obstruction.

FIGURE VI. Case VI. Showing the degree of the obstruction as indicated by the degree of distension of the caecum.

radiographic examination revealed the bowel lumen to be clear and the colostomy was allowed to close (see Figure III).

The patient has remained well, the bowels acting normally.

The interesting feature in this case was the extent of the filling defect in the colon. This long filling defect is seen in many cases of carcinoma in which associated diverticula may also be seen in the skiagram. The long filling defect associated with a short history and a mild inflammatory reaction was a ray of hope which could not be overlooked, and the simple nature of the condition was eventually established.

FIGURE VII. Case VI. Showing the lumen of the bowel patent and the presence of diverticula. The loop of bowel which was allowed to retract back into the abdomen is evident on the right of the figure.

CASE V.—The patient, A.D.H., male, aged sixty years, was first seen on May 23, 1936, complaining of abdominal pain for about three weeks.

The temperature was 36·9° C. (98·4° F.) and the pulse rate 76. On examination, a large mass was palpable in the abdomen in the region of the umbilicus. An opaque enema examination by Dr. Praagst revealed a large, irregular filling defect in the transverse colon extending from a point slightly to the right of the middle line for a distance of ten centimetres (four inches) (see Figure IV).

A diagnosis of carcinoma of the transverse colon was made.

At this stage, although well acquainted with cellulitis and inflammatory lesions in the sigmoid colon, I had not encountered such a condition in the transverse colon, and again mistook at operation an inflammatory lesion for a malignant one. An anastomosis between the transverse colon and sigmoid, thought to be palliative, resulted in the regaining of more weight than was lost, the complete disappearance of the lump clinically and perfect health.

This lesion occurred further to the right than any others I have encountered of the condition, and is only included for that reason.

CASE VI.—The patient, J.W.R., female, aged seventy-three years, was first seen on July 10, 1937. This case is only included to show the extent of the dilatation of the large bowel that can occur in these cases (see Figures V, VI and VII).

This patient obtained relief of her obstructive symptoms naturally when waiting twenty-four hours to have the loop of bowel withdrawn for colostomy opened. The rod was withdrawn and the bowel allowed to retract. When deflation had occurred further radiographic examination showed diverticulosis of the sigmoid colon and no obstruction. The patient meantime remained perfectly well.

It is obvious that the diagnoses in Cases V and VI are open to doubt on the score that the condition may have been malignant, but malignant obstruction seldom disappears. Malignant tissue may grow slowly, but the obstruction that results is progressive and does not disappear for months either clinically or radiographically.

Whether or not the condition of cellulitis of the colon is always associated with diverticulosis I am not sure. In this series, in all except Case V, diverticula were proved to be present. There would seem to be no reason why such should be the case, as any focus or abrasion would suffice for the origin of a cellulitis, but it would seem that with this condition diverticulosis is common.

This series of cases, which are not consecutive and are only a few of the cases of diverticulitis with cellulitis that I have encountered, is presented because in them the intraperitoneal complications were absent or insignificant and the cellulitic and obstructive signs outstanding. Although I have seen many other cases of diverticulitis with mixed peritoneal and cellulitic reactions, it is from the cases presented that, rightly or not, the clinical picture presented has been visualized.

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ANÆSTHESIA IN GALL-BLADDER DISEASE: METHODS OF TREATMENT IN THE SO-CALLED "BAD RISK CASES".

By Harry J. Daly,
Sydney.

It should be remembered in considering anæsthesia for surgical diseases of the gall-bladder that the pathological changes present in this organ are often accompanied by disease of the liver, bile-ducts and pancreas. Abnormal increase in blood pressure and signs of cardiac failure are quite commonly seen in patients over fifty who have had cholecystitis with calculus formation for some years. Such complications must be investigated and suitable treatment carried out before these patients are fit to take an anæsthetic.

Hepatic Insufficiency.

Present-day knowledge of the functions of the liver in health and disease is indefinite. We know, however, that the liver plays an important part in the disposal of poisons, such as anæsthetic agents. We also know that the liver stores glycogen and can metabolize carbohydrates. Davis and Whipple,[1] of America, proved that starved animals were very susceptible to liver injury by anæsthetics, and that animals fed on a diet rich in carbohydrates and sugar, for some days preceding anæsthesia, were able to protect themselves from liver damage. In cases of suspected liver damage, therefore, a liberal supply of carbohydrates and sugar before anæsthesia is a valuable safeguard.

Some anæsthetics cause serious liver damage. This is especially true of chloroform. Dr. Wesley Bourne,[2] of Montreal, who is an authority on anæsthetics and their effects on liver function, states that chloroform poisoning is synonymous with chloroform anæsthesia. By means of bromsulphthalein dye retention tests he found that after ether anæsthesia liver function as a rule had returned to normal next day, whereas after chloroform anæsthesia liver damage, as evidenced by dye retention in the blood, continued to progress and did not reach its maximum until next day. The liver took eight days to return to normal after half an hour of chloroform anæsthesia. These facts are mentioned as a warning against the use of chloroform if impairment of liver function is suspected.

Pancreatic Disease.

That the pancreas is often involved in biliary tract disease is shown by the frequency of glycosuria in this class of patient. If on investigation this is proved to be due to true diabetes, then before any anæsthetic is given it must be brought under control by suitable treatment. An anæsthetic which

has a minimal effect on metabolism should be chosen. Spinal anæsthesia or local anæsthesia plus one of the gaseous anæsthetics might be the most suitable form of anæsthesia.

CARDIOVASCULAR DISEASE.

In some cases in which the hepatic or pancreatic function is little, if at all, disturbed, the deciding factor in choosing a suitable anæsthetic agent may be disease of the cardiovascular system. Arteriosclerosis is a frequent accompaniment of disease of the gall-bladder, and this fact is borne out by the large number of patients who show abnormally high blood pressure and thickened blood vessels. Many of these patients are on the verge of hypertensive cardiac failure, and electrocardiographic studies may be of value in cases of cardiac irregularity or of suspected coronary occlusion, and give an indication of the need for pre-operative treatment. Any complaint of weakness, undue fatigue, palpitation, dyspnœa or chest pain on ordinary activity should be investigated. Patients suffering from cardiac decompensation with dyspnœa and œdema which is not responding to treatment, and also patients with severe anginal symptoms or recent coronary occlusion, must be considered bad anæsthetic risks. Sir James Mackenzie's dictum, "that the subjective symptoms of heart failure are never absent when the heart's efficiency is in any way impaired", is a very good rule by which to be guided, and it is surprising how well in some cases the heart can function in the presence of high blood pressure. High blood pressure in itself does not contraindicate inhalational anæsthesia. The ability of the heart and the circulation to respond to rest in bed and general treatment is astounding, so that many of these patients, who at first sight appear desperate anæsthetic and surgical risks, become reasonably good risks under treatment. The benefits of pre-operative rest and care were forcibly brought home to me by the improvement effected, by this means, in many old men suffering from prostatic disease under the care of the late Dr. Harry Harris. It was the rule rather than the exception to find arteriosclerosis and atheroma with cardiac failure in these patients, and it was a routine procedure to investigate all phases of their illness. They were kept under observation, with rest and suitable treatment, for weeks before any operative procedure was carried out. The secret of Harris's success was the utmost attention to this pre-operative management, which made these patients reasonably good anæsthetic risks.

PREPARATION OF THE "BAD RISK" PATIENT FOR ANÆSTHESIA.

In the pre-operative care, to ensure against liver injury and acidosis following anæsthesia, all patients, especially those who are "bad risks", need a liberal supply of carbohydrate in the form of glucose. A good preparation easily taken is "Glucose D".[3] Some authorities give as much as 300 grammes every twenty-four hours till the liver function shows improvement as indicated by certain dye tests. If glucose cannot be tolerated by mouth, then glucose (500 cubic centimetres of a 10% solution) may be given intravenously till tests show that the liver is functioning satisfactorily. Patients showing jaundice should be given calcium gluconate 0·3 gramme (five grains) thrice daily intravenously for some days to correct any tendency to oozing during operation. This is important if oxygen is being used with the anæsthetic,

because I am of the opinion that too liberal a supply of oxygen often causes oozing during operation. This oozing ceases, however, when the anæsthetic is discontinued. Copious fluids should be given to improve liver function, to combat dehydration, to dilute toxins in the blood and to bring about their elimination by the kidneys. Regulation of the bowels should be undertaken, but drastic purgation avoided. If anæmia is suspected, a blood count should be carried out, and if the hæmoglobin value is down to 60% the question of blood transfusion before operation should be considered. Disease of the lungs must receive careful consideration and treatment, as any chronic pulmonary disease may prohibit the use of an otherwise suitable inhalational anæsthetic.

It is when these aspects of the patient's condition have received attention that the question of the selection of the most suitable anæsthetic for the individual patient comes up. This should be determined by consultation between the anæsthetist and the surgeon. Routine anæsthesia does not take care of the "bad risk", and though unusual anæsthesia may be tiresome, it is at times necessary if a patient is to be tided over a critical operation.

The need for muscular relaxation in biliary tract surgery is obvious, yet to bring the "bad risk" patient down to deep third-plane anæsthesia reduces the margin of safety. Muscular relaxation facilitates exposure, which again shortens the time of operation. A pillow under the knees combined with raising the head is a help. The position of the gall-bladder rack appears at times to be too high, and this interferes with relaxation, pushing the abdominal contents up into the wound and interfering with free respiration.

Two "Bad Risk" Types Considered.

Now let us consider two types of patient: the first, a difficult surgical and bad anæsthetic risk, and the second, often a desperate anæsthetic risk.

The first type is the short, stocky, fat patient, a smoker and drinker, who is often a bad risk because the operation takes half as long again as it normally would and because he must be kept under deep anæsthesia for the surgeon to work at all. The danger in store for this patient is post-operative respiratory complications, which have a direct relationship to the time he has been under the anæsthetic. This time factor and its bearing on respiratory complications have been clearly shown by a series of carefully worked out statistics at Water's anæsthetic department in Wisconsin University Hospital, Madison, the United States of America. If in such a case inhalation anæsthesia is chosen, lack of oxygen as evidenced by cyanosis must be overcome at all costs, if necessary by the additional administration of oxygen. No degree of obstruction of the airway is to be permitted. The best way to ensure this is to give a small dose of morphine sulphate, 0·01 gramme (one-sixth of a grain), and a large dose of atropine sulphate, 0·0008 gramme (one-seventy-fifth of a grain), and when the patient is anæsthetized, to pass a large Magill type endotracheal tube. By this means a good airway is secured and the vocal cords lie flaccid and abducted. Should respiratory failure threaten, no better means of artificial respiration could be at hand. However, I have never seen a patient stop breathing when such a tube is in place. Relaxation of the abdominal muscles is never complete with inhalational anæsthesia until the vocal cords are abducted and paralysed.

It should be the anæsthetist's aim to have this patient leave the table dry, warm and pink, with free, easy respiratory movements, and not sweating, cyanosed and cold, with respiration interfered with by mucous inundation of the lungs and air passages. Atropine is a valuable aid in preventing the mucous inundation so often seen with ether anæsthesia. When the operation is completed, the elimination of the anæsthetic should be facilitated by the insertion of a large artificial airway, and, if necessary, by the administration of carbon dioxide and oxygen. By such means post-operative lung complications are often avoided.

Many surgeons, especially in America, advocate spinal anæsthesia for such a stocky, muscular patient, some of its advantages being complete muscular relaxation—cadaveric, they call it—enabling the freest exposure of the operative field, and thus rendering a difficult operation easy. Spinal anæsthesia has no effect on the liver or on metabolism generally. Unfortunately, respiratory complications occur almost as frequently after spinal as after general anæsthesia. In addition, high spinal anæsthesia carries *per se* an added risk of respiratory paralysis and shock. The vomiting which may occur following the fall of blood pressure and the handling of the upper abdominal organs is disturbing. With "Novocain" spinal anæsthesia one can only be sure of about fifty minutes of anæsthesia. With "Percaine", on the other hand, at least two hours may be secured; but, in any case, the patient becomes very weary and at times restless after forty minutes of spinal anæsthesia.

The second type is the emaciated, dehydrated patient, who has had many attacks of gall-stone colic, till finally a stone has lodged in the common duct, causing deep jaundice. Nausea and vomiting are often present, and only liquids can be taken. Signs of acute infection of the biliary tract and empyema of the gall-bladder may be superadded. Continuous intravenous glucose and saline infusions are called for, both to dilute the toxic products in the blood and to restore liver function. The intravenous administration of calcium is indicated to combat oozing at operation. Such patients are desperate risks, both from surgical and anæsthetic viewpoints. What anæsthetic are we to use here? Operation may be performed under local anæsthesia, but this is often unsatisfactory. If for any reason ether must be used, anæsthesia should be maintained in as light a stage as possible and oxygen freely given. Nitrous oxide and oxygen anæsthesia is not satisfactory by itself because of the difficulty of securing relaxation without some degree of oxygen want; however, the addition of a small amount of ether will bring about sufficient relaxation and thus the oxygen can be increased.

ANÆSTHETIC METHODS.

The best method of dealing with these "bad risk" gall-bladder patients, and one which in my opinion gives the most satisfactory results, is to be found in the closed carbon dioxide absorption technique. I first saw this method of anæsthesia used in many clinics in America in 1935, and I was so impressed with its advantages, especially for bad risk patients, that I brought a machine back with me. Nitrous oxide and oxygen may be used in this manner, and the addition of a few cubic centimetres of ether into the closed system will bring about sufficient relaxation. However, a new gas, cyclopropane, has many advantages over any other inhalation anæsthetic

for bad and desperate risks. This is a powerful gas, which produces anæsthesia and relaxation in the presence of, and diluted by, high percentages of oxygen. The main features of the closed method are that the gases are made to circulate through soda-lime, which removes the exhaled carbon dioxide. Theoretically, once anæsthetic equilibrium in the blood is established, no more gas need be added, but oxygen sufficient for the metabolic needs must be given at the rate of 250 to 300 cubic centimetres per minute. Practically, it is difficult to maintain a completely gas-tight system, and a small trickle of gas has to be added to make up for leaks. The main feature of the closed technique is that it protects the patient from muscular exertion and fatigue during anæsthesia. Respiration is exceptionally easy, quiet and under control. As the carbon dioxide is absorbed by the soda-lime, a certain amount of heat is produced in the closed system. This is an advantage, as the patient breathes only warmed gases. This saves the patient from heat loss, whilst water loss is also controlled, as in this system the gases are saturated after the first few breaths. Sweating is rarely observed, because the patient is not exerting himself, that is, he is not stimulated by carbon dioxide retention and not panting to gain oxygen. Loss of fluid and loss of bodily heat are the forerunners of shock, and these losses are avoided in a closed system.

In conclusion, I feel convinced that the closed carbon dioxide absorption method, with the use of cyclopropane and oxygen, or cyclopropane as an adjuvant to nitrous oxide and oxygen, offers the best solution of dealing with the bad gall-bladder risk. Unfortunately the apparatus is expensive and is not yet in common use in this country, though it is available in all the large centres in America.

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MOTOR MECHANISMS OF THE FOREBRAIN.[1]

By F. GOLDBY,
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IN choosing a subject that is as much concerned with function as it is with structure, it may seem to you that I am trespassing rather far from my legitimate field as an anatomist, so perhaps I should begin by making an attempt to justify my choice.

One of the functions of an anatomist is to describe the structure of the body so as to provide a foundation on which the physiologist and the clinician can build. This is not his only function, nor to my mind his most important one. Anatomy is a branch of biological science. It is distinguished from physiology or clinical medicine and surgery not by the subject that it studies, but by the technique that it employs. Any biological problem can be attacked from three angles at least: the clinical, the physiological and the anatomical. Each worker brings his own particular technique to bear on it; a solution will be reached only by combining the efforts of all three. It is impossible to narrow the field of anatomy down to an exclusive interest in structure for its own sake without grave loss, and in no part of the body is this more obvious than in the central nervous system. It has, indeed, proved a happy hunting ground for those who wish to pick up numberless structural facts, and the volume of these collected in anatomical literature is prodigious and overwhelming. It is only when an anatomist ceases to be merely a collector, and directs his activities in an attack on specific biological problems, that his work becomes worthy to be called a science.

Movement, or more generally motor behaviour, is one of the most characteristic of the attributes of animal bodies. It constitutes a biological problem in which all of us, whether as surgeons or physicians or biologists, must be interested. Underlying its manifestations in man and all the higher animals is a complex mechanism in which the brain, the peripheral nerves and the muscles all play a part. On the muscles and the peripheral nerves much, but by no means all, of the anatomist's work has already been done. On the basis of this work the physiologists and the clinicians have been able to apply their own particular techniques, and often with strikingly successful results. In the brain the position is quite different; so far from the anatomist becoming a physiologist, one may say that the physiologist becomes an anatomist whenever he turns his attention to this part of the body. The conduction of a nervous impulse in a single nerve fibre we can already think of in terms of the electrical or other phenomena that accompany it, but in the brain, and especially in the forebrain, we cannot yet think in other terms than those of anatomical structure, with anatomical connexions and relationships, both intrinsic and extrinsic. For this reason the forebrain

[1] Paper read to the Royal Australasian College of Surgeons at the South Australian annual meeting held in Adelaide on Tuesday, November 2, 1937.

lies peculiarly in the province of the anatomist, and the problem of its relationship to motor behaviour is as clearly anatomical as it is physiological or clinical.

It is known, mainly from clinical and pathological observation, that the two gross subdivisions of the forebrain, the cortex and the basal ganglia, are both related to motor function. Structurally they are almost the antithesis of one another. The cortex consists of a superficial laminated arrangement of neurones, extensive in area. It shows marked complexity of structure and an intrinsic differentiation into many different parts. The basal ganglia are buried deeply and are extensive in bulk. Apart from the thalamus they are not histologically complex, and they show little intrinsic differentiation. The *corpus striatum,* with which I shall be mainly concerned, contains only two types of neurone: a small type, with a fine axone which ends inside the *corpus striatum,* and a large type, with a longer axone, which ends in structures outside. The larger neurones are found mainly in the *globus pallidus,* the small ones in the putamen and the caudate nucleus. The putamen-caudate mass can be looked upon as a single structure. Its subdivision, in any case not complete, has been brought about secondarily by the passage of the fibres of the internal capsule through it, and these fibres have for the most part no direct connexion with the *corpus striatum.*

Striatal and cortical lesions lead to motor disturbances that differ markedly in character. For the moment it will be convenient to limit ourselves to the well-known motor cortical area, the Area 4 of Brodmann. Foerster (1936) has defined some of its functional characteristics in man very clearly. It is concerned with the localized innervation of small muscle groups, moving single segments of a limb. Its action is rapid and precise. It is phasic, in contrast to the persistent tonic postural activities of the brain stem. It is better developed in relation to the distal than to the proximal segments of the limbs and the trunk, and more to the flexor than to the extensor groups of muscles. In rats, where cortical mechanisms generally are of far less importance than in Primates, it has been shown by Barron (1934) that three weeks after pyramidal section the animal is normal to superficial observation. On closer observation it is found that there is some deficiency of the flexor movements of the digits of the forelimb, and this deficiency is permanent. The animal, for example, always holds food in the hand on the normal side, although before the operation both hands were used indiscriminately.

The loss of these finer movements makes up the negative side of the symptomatology of lesions of the motor cortex. On the positive side, disorders of tone and posture make their appearance in man, although they are not so persistent or invariable as the negative symptoms. These positive symptoms are due, as Hughlings Jackson was the first to suggest, to the excessive and disordered activity of lower mechanisms which have escaped from normal cortical control. They tend to produce an exaggerated standing posture, in which extensor predominates over flexor tone. The hemiplegic attitude does not appear, however, to be a complete "decerebrate rigidity"; the arm usually takes up a position of semi-flexion and pronation. Blair and McDowall (1933) have shown that this same posture, with partial flexion of the forelimb and extension of the hindlimb, may be obtained in the cat after lesions confined to the Area 4 of the cortex. This flexor attitude of the forelimb seems

to depend on the integrity of adjacent cortical areas, since it can be abolished by extending the original lesion; it provides an example of extrapyramidal motor activity. The mechanism, therefore, that lies behind the attitude of the hemiplegic may be more complex than we thought, involving the activity of other cortical areas spared by the lesion.

The structural counterparts of these functions of the motor cortex are in one respect simple. The innervation of muscles is brought about through the axones of the Betz cells, which form a long descending pathway which is connected directly or almost directly with the motor nuclei of the lowest level, the anterior horn cells of the spinal cord. It connects, in other words, almost directly with the "final common path". The presence of this comparatively simple tract does not, however, explain all the aspects of the activity of the motor cortex. It is known in animals, and it has been particularly clearly demonstrated by Tower (1935) in the cat, that section of the pyramidal tract in the medulla, while it causes defects mainly of flexor movements, much as would be expected, does not lead to any significant increase of extensor tone. This appears only when, in addition to section of the pyramid, the corresponding motor cortex is removed. The control of postural tone, exerted by Area 4, must be effected through tracts other than the pyramidal, or perhaps through collateral branches of the pyramidal fibres that arise above the medulla. The removal of pyramidal influence from the anterior horn cells is not enough to allow the escape of postural mechanisms; this escape must occur at a higher level, almost certainly in the brain stem. Either the pyramidal tract is more complex than we supposed, or the motor cortex must make use of extrapyramidal projection tracts in the performance of some of its functions.

Having stressed these few aspects of the structure and function of the motor cortex, I must pass on to the consideration of other regions of the forebrain; but I should like to pause before doing so to point out the character of the work that has been responsible for progress in the particular field that we have been considering. It is all based upon the anatomical work of men like Betz, the physiological researches of Ferrier and Sherrington, and the clinical observation of Hughlings Jackson, to mention only a few names. It is a good example of the fruitful co-operation between the clinician and the laboratory worker, in which it would be difficult to assign to one or the other the predominant rôle. Purely structural or purely functional studies could never have led to the body of knowledge we now possess.

Turning to the *corpus striatum*, we find that the position is not quite the same. Because of its situation it is a structure difficult to approach experimentally, and it is not responsive to electrical stimulation. Lesions in it give rise to little in the way of motor disturbance that can be observed directly in an animal which cannot describe its feelings or disabilities. Even when motor disturbance is evident, it may be due to damage to the internal capsule or the subthalamus, structures which are very intimately related to the *corpus striatum*. Uncomplicated lesions are difficult to produce experimentally. It is, therefore, not surprising that most of our knowledge has come from the clinician rather than from the laboratory worker. The clinician has the advantage of studying the experimental lesions of Nature, sometimes, in the case of the *corpus striatum*, strictly limited to that organ, and occurring in a patient both willing and anxious to describe his symptoms.

The name of Kinnier Wilson is prominent in this particular field, and much of what I have to say will be based upon his work.

In disease confined to the *corpus striatum* the lesion is always of a destructive character. Symptoms, therefore, must be entirely of a negative kind, due to the loss of striatal influence on other parts of the nervous system, and not directly to the activity of the *corpus striatum* itself. These symptoms have been defined by Kinnier Wilson in three categories: dystonia, the appearance of involuntary movements, and the absence of true paralysis. There are, it is true, some observations that suggest that in those cases where choreic or athetoid movements predominate, the *globus pallidus,* with the main efferent tracts of the *corpus striatum,* is spared. The destruction is practically confined to the putamen and the caudate nucleus, while if it extends to the *globus pallidus,* tremor and excessive tone form the most striking features of the clinical picture. Using a kind of terminology in common use, it is tempting to see in the *globus pallidus* a centre for the initiation of movement, normally inhibited by the putamen-caudate, and in the whole *corpus striatum* a centre for the control of tone in general. The *globus pallidus* becomes the motor centre of the *corpus striatum,* related to the putamen-caudate much as the Area 4 of the cortex is related to the associational cortical areas.

I think that in these attempts to make a precise functional localization in the *corpus striatum* we have been over-confident, led on perhaps by success in other parts of the nervous system. It was inevitable, but a little unfortunate, that the first motor part of the forebrain to be investigated by precise methods was the cortical Area 4. Immediate success in this particular area has led us to hope that similar results may be obtained elsewhere, and unconsciously we begin to think of motor function in terms of strictly localized centres. The very term "centre" is a source of loose thinking. If the term has any concrete meaning it should be applied to some definitely localized mass of neurones which exercises exclusive control over some equally well defined activity. The absurdity of the conception becomes obvious as soon as it is stated in this way. No such structure has ever been defined anatomically. Respiration, for example, is said to be presided over by a "centre". Behind the process of respiration there is a very complex neural mechanism, involving the fibres of many cranial and spinal nerves and their nuclei, and between its afferent and efferent sides a complex network of interconnexions. This mechanism can be interfered with at many points, and by the term "respiratory centre" we mean no more than a particularly vulnerable region in the network. It so happens that such a region in the respiratory mechanism lies in the *medulla oblongata.* By very crude transection experiments this can be shown fairly definitely, but no one collection of neurones has ever been defined anatomically as having exclusive control over the whole mechanism. Nor from the nature of the case is any such collection likely to be defined. In short, the term "centre" is essentially physiological, and cannot be translated into precise anatomical terms. The same statement would apply to what appear much better defined "centres" in the motor cortex. The area, for instance, from which movements of the hand may be elicited, is no more than one link in a complex mechanism that includes transcortical connexions, basal ganglia and nuclei in the brain stem and spinal cord in its ramifications.

Much of this is no doubt obvious, but it seems worth stating, for consciously or unconsciously, when we think in terms of centres, it is difficult not to visualize the nervous system as a collection of discrete anatomical entities, each with a defined function, and all forming together a mosaic of more or less independent parts. It is needless to stress how inadequate such a conception is in face of the varied and confusing symptom complexes that accompany lesions in the nervous system. It is particularly misleading in relation to the *corpus striatum*. If this is the centre for certain functions, one should at least be able to define the loss when it is damaged. It is, however, very difficult to do so. Sensation and intelligence are not impaired, and there is no actual paralysis. There is no movement which the patient cannot perform, although he actually performs all movements badly. In nearly every one of his activities the patient is not paralysed, but hampered by the excessive tone or tremor or both, which affect his musculature generally. It is surely clear that we are dealing with a system that has been damaged and is functioning inefficiently as a result, not with a centre in which certain functions are localized and which has been lost. The whole motor system has been thrown out of balance by the lack of certain impulses that in the normal person are poured into it by the *corpus striatum*.

Neither dystonia nor the development of tremor or of choreic or athetoid movements is found exclusively in striatal lesions. The integrity of the *globus pallidus* may be necessary if the choreic or athetoid movements are to appear, but it also seems that parts at least of the cortex must be capable of function. Surgical removal of that part of the motor cortex that corresponds to the part of the body affected will sometimes lead to the cessation of the movements. They are not exclusively dependent on the *globus pallidus*, and to say, as has been said, that after destruction of the putamen-caudate the *globus pallidus* is left free to exhibit its own primitive form of activity as choreic or athetoid movements, is as much at variance with clinical and pathological observation as it is in my opinion with the facts of comparative anatomy and the judgement of common sense.

Both structurally and functionally the *corpus striatum* must be studied as part of a complex motor system, which includes the cortex as well as lower centres. Its study is a difficult problem, particularly in the complex nervous system of man, but an artificial over-simplification will not help us. There is, however, a natural simplification that is available. We may study the origins of these structures and their interrelationships, tracing their development from simpler beginnings in the reasonable hope that they may throw light on the more complex endings. It is in the development of a technique for the study of growth and differentiation that the anatomist has made his greatest contributions to the understanding of biological problems. The technique may be embryological or it may follow the methods of comparative anatomy. It is the latter method on which I now intend to dwell for a short time.

The first broad fact of importance is that the cerebral cortex, so far as those parts that are not mainly olfactory in function are concerned, is almost exclusively a mammalian structure. In all submammalian vertebrates this cortex is either vestigial or rudimentary. In the more generalized and primitive animals, like some of the living amphibia, it is present, but is small in extent and almost entirely undifferentiated. In some, for example,

the cortex of the adult animal is very like that of the human embryo during the fifth week of its development. In the reptiles there is a little differentiation, to the extent that some anatomists have claimed to recognize at least a primordium of the mammalian non-olfactory cortex, and it was for this reason that I undertook an investigation of the forebrain of the lizard. The reptilian cortex will serve as a point of departure from which we can follow the main line of cortical evolution.

In the reptile no evidence can be found for the presence of descending projection tracts in any way comparable with the pyramidal tracts of mammals. The only efferent connexions of the cortex that can be identified with even approximate certainty are short and end in the *corpus striatum* or other basal structures. Cortical impulses can therefore affect the motor nuclei of the brain stem and cord only indirectly. There is no direct connexion from the cortex to the final common path as in mammals. Further, the reptilian cortex is electrically inexcitable (there is some conflict of opinion on this point), and quite extensive lesions produce no obvious motor defects. There is, in effect, no evidence for any localization of function in this cortex, nor of any precise control of movement or posture. It probably exerts a general influence on other neural mechanisms, at times facilitating and at times inhibiting their activity. There is some evidence for this, but there is no need to recapitulate it here.

Even in primitive mammals a marked advance in cortical evolution has taken place. The cortex is more extensive and more complex histologically, and the well-known primary projection areas have appeared. These findings are anatomical; physiological advance is not so marked. In most of the simpler mammals the motor area, as defined by electrical stimulation, is not clear-cut. Movements can be obtained from the facial and jaw muscles, from the neck and from the forelimb, but less easily and constantly from the trunk or hindlimb. Destruction of this cortical area leads to some defect in the finer flexor movements of the forelimb, as in Barron's experiments on the rat, but the defect is compensated fairly rapidly. It is never very conspicuous and is not accompanied by any disorder of tone. Similar results have been obtained in many mammals, and it has been found that in some of them the pyramidal tract is small and can be traced no further than the cervical part of the spinal cord.

In Primates, and particularly in man, the motor cortex has become more extensive and much more clearly defined histologically and functionally. The pyramidal tracts are proportionately larger; they account for about 7% of the white matter in the spinal cord of the dog and about 40% in man. They extend throughout the whole length of the cord. Physiological localization, as shown by Foerster in man, has become exceedingly precise, and damage, even to quite small parts of the motor cortex, shows itself in marked loss of movement and disorder of tone.

These results of damage to the pyramidal system in man are very striking, and contrast strongly with the results of similar lesions in most other mammals, and also with the results of quite large extrapyramidal cortical lesions in man. The latter may be characterized by slight and transient motor symptoms, but often by none at all, and it is interesting to speculate why the pyramidal tract, of all the efferent projection tracts of

the cortex, should have risen to such a position of dominance in the human motor systems.

The pyramidal tract arises in close association with a sensory projection area of a special kind. It is the somæsthetic sensory area, concerned with sensation from the skin and probably with proprioceptive sensibility as well. As far as the lower mammals are concerned, it seems probable that skin sensation is most highly developed on the snout and face, equipped as this region is with very sensitive vibrissæ. It is certainly this region that is most freely used in exploring the environment, guided always by the sense of smell. Second perhaps in importance are sensory impressions from the forelimbs, which are used for grasping and holding food and carrying it to the mouth. Although direct anatomical evidence is lacking, it seems likely that sensations from these particular areas of skin would be the first and perhaps the only aspects of skin sensibility to gain representation in a somæsthetic cortical area in primitive mammals. If this is so, it is no coincidence that the efferent projection tract related to this area should acquire control over the movements of the face, jaws and forelimb. It is movements of just these parts that can be elicited most easily from the motor cortex of primitive mammals.

The characteristics of the actual movements are also peculiar. Movements of the snout and vibrissæ in the search for food, movements of the hands in grasping it and of the jaws in eating it are essentially non-postural. In the case of the forelimbs use is made mainly of the flexor muscles, and in general of smaller rather than larger muscle groups. The movements are phasic, with none of the characteristics of tonic contractions; in fact, for their execution tonic contractions must be inhibited. They are also less stereotyped; they must be capable of rapid variation to meet the varied conditions incidental to the search for food and its prehension. No subcortical neural mechanisms exist which can cope with motor activity of this kind. There is some evidence, which I will discuss later, that the *corpus striatum* in some animals is capable of integrating a complex behaviour pattern, but however complex it is invariable. Structures in the subthalamus may integrate rhythmical activities like walking, but these are of an obviously different character, while the brain stem is taken up very largely with postural reflexes, again different from the movements we are considering. It seems, therefore, once more hardly a coincidence that the pyramidal tract, the projection tract of the somæsthetic area, should pass by the *corpus striatum* and the mechanisms of the mid-brain and medulla, and connect directly, or almost directly, with the motor nuclei of the lowest level, the final common path of all efferent impulses from the nervous system. Here it seems we have the crux of the matter. The cortex, through the pyramidal tract, has taken control of the last link in the motor chain, and through it can exercise a far more detailed control of movement, a control that can express that characteristic property of the cortex, its plasticity and power to vary its response to present stimuli in the light of others previously received.

Why, then, has the pyramidal tract extended its field of control in the Primates and in some other mammals? Wood Jones has stressed the importance of the arboreal habit as a factor in the evolution of the Primates and man. This habit necessitates the use of the hindlimb equally with the forelimb in grasping, in phasic movements of the small muscles of the distal

segment of the extremity, and it is just this kind of movement that the pyramidal tract is adapted to serve. It seems at least possible that extension of pyramidal control is related to the arboreal habit in this particular case.

We are now in a position to visualize at least the broad outlines of cortical evolution so far as it concerns the pyramidal system. At first, in amphibia and reptiles, the cortex has no precise control of movement. It yields no evidence of localization of function, but probably has some general effect of facilitating or inhibiting mechanisms in lower levels of the nervous system. The first step towards the mammalian condition was probably taken when specific sensations acquired localized representation in the cortex, reaching it by way of the thalamic radiations. From the somæsthetic area thus developed, which may at first have received sensation only from the face and forelimb, the efferent projection tracts acquire direct control over the motor nuclei of the muscles of the anterior half of the body. This pyramidal tract serves a new type of movement which I have just described. Since this type of movement is in primitive mammals limited to small parts of the body, it needs little in the way of postural inhibition or adjustment, so that destruction of the pyramidal tract or of the cortex from which it arises leads to insignificant disturbances in the distribution of tone. Similarly, because the particular phasic movements served by the pyramidal tract play a comparatively small part in the total behaviour of these animals, especially in captivity, the actual paralysis or loss of movement is not very striking.

The higher mammals show an extension of control from the motor cortex to all parts of the body. This extension is quite marked in the carnivora, but most of all in the Primates and man. Movements characteristic of pyramidal activity come to play a very large part in the animal's total behaviour, and the loss, after damage to the system, is correspondingly greater. In order that these extensive cortical motor activities shall have free play, more and more inhibition of postural and tonic reflexes becomes necessary. The tonic and postural mechanisms become more and more interwoven with the pyramidal system as a result, so that damage to this system leads now not only to widespread paralysis, but also to grave disorders of posture and tone.

No account of the development of the motor functions of the cortex can be even approximately complete without some mention of the extrapyramidal motor areas. These have been defined in man by Foerster (1931) and have recently been investigated in considerable detail by Tower (1936) in the cat. Foerster has shown that there are fairly large areas in each of the lobes of the hemisphere from which characteristic "adversive" movements can be obtained by electrical stimulation. These are essentially mass movements, usually contralateral, consisting of a rotation of the trunk with coordinated movements of flexion and extension of both limbs. Less generally coordinated movements of the eyes and of the mouth and lips are obtained. The adversive movements are the most characteristic of the responses obtained from extrapyramidal fields. They develop slowly and outlast the period of stimulation, and differ in almost every way from the discrete phasic responses from the Area 4. Tower's important work has carried the analysis of extrapyramidal function in the cat much further. Only a few of her results can be discussed here. Besides initiating the adversive movements already described, there are in the cat certain areas which are able to initiate

simple rhythmic activities, of which walking is one. These rhythmic activities can also be inhibited from extrapyramidal areas, stopping the animal, as it were, in a posture of attention. Tone, particularly in the extensor muscles, can also be inhibited from the cortex. On the anatomical side, Tower has found that the areas from which these activities can be obtained, although capable in many cases of fairly exact definition, do not as a rule coincide with defined cytoarchitectonic areas; they often overlap each other and even extend into the Area 4. Some of the extrapyramidal functions are so widely spread as to exhibit almost no localization at all, and in some cases similar activities may be elicited from widely separated parts of the cortex. There is also evidence that, although the extrapyramidal functions have their own projection tracts independent of the pyramids, these are short and extend no further than the basal ganglia or mid-brain. They have no direct connexion with the final common path.

It is clear that in their lack of precise definition, in their short projection fibres running to the basal ganglia and mid-brain, and in their generalized function of inhibiting or facilitating complete patterns of movement, some of the extrapyramidal aspects of the mammalian cortex resemble very closely the whole of the cortex of reptiles and amphibia. They are genuinely primitive aspects of cortical structure and activity, carried over from a pre-mammalian ancestry. Strict localization of function is not a primitive characteristic of the cortex; this is primitively a structure equipotential in all its parts, to use Lashley's expression, on which both sensory and motor localization are subsequently impressed. Such localization is only incipient in the lower mammals, and it has risen to a high degree only in one group, the Primates.

These observations on extrapyramidal structure and function have some bearing on Lashley's work on the function of the cortex in the rat, neurologically a primitive mammal. He has found that removal of up to about 50% of the rat's cortex not only has little effect on its ordinary activity, but little even on its capacity for learning and retaining certain habits or tricks, each of which requires the synthesis of a number of movements into a regular sequence. This synthesis can apparently be effected at subcortical levels. The presence of cortex is essential, but it is the total mass and not any one localized part that is important. Less than about 50% of the amount normally present is inadequate. In Primates these generalized functions are still present, but they have fallen into the background, as it were, being overshadowed by the functions with a strict localization that have risen to eminence. They represent, however, the anatomical and physiological counterpart of the clinical observation that cortical lesions outside the Area 4 give rise either to slight and easily compensated motor defects or to none at all. It is not that motor function is lacking, but that it is widespread, often represented in more than one part of the hemisphere, and that it works mainly by facilitating or inhibiting lower mechanisms that have some power of independent activity.

There remains the *corpus striatum* to be considered. In the primitive amphibia it is a poorly differentiated structure like the cortex, connected, by fibres that are probably mainly afferent, to the thalamus, which shows a similar lack of differentiation. It appears to discharge mainly into the subthalamus and the tegmentum of the mid-brain. In the reptiles the basal ganglia, unlike the cortex, show a marked increase both in size and degree

of differentiation. The *corpus striatum* indeed forms the largest part of the forebrain, and several distinct parts can be recognized in it. In general, its connexions are still the same, but they are much more abundant. The afferent and efferent fibres of the striatum now form a system comparable in relative size to the internal capsule of mammals. It is obvious that the striatum must play a large part in the forebrain activities of reptiles, but exactly what that part is, is difficult to determine. Electrically it is inexcitable, and unilateral lesions lead to no obvious motor disability. Extensive bilateral lesions, on the other hand (which amount practically to complete decerebration, since the cortex is inevitably destroyed), leave the animal extremely sluggish and apparently disinclined for movement. It is only with difficulty and by the use of gross sensory stimuli that it can be induced to move at all, but then the movements are normal and there seem to be none of which it is actually incapable. Left alone, the animal tends to adopt a posture of slight hyperextension, faintly resembling extensor rigidity. Beyond this no deficiency is observed except that the animal is never seen to feed spontaneously. The jaw movements and the swallowing reflexes are normal, but they are never combined into the proper sequence necessary for spontaneous feeding.

This last observation would not have been considered important had it not been for a more extensive series of experiments made by Rogers (1922) on birds. In the bird the *corpus striatum* constitutes practically the whole hemisphere, and is far more complex in its differentiations than in the lizard. The cortex, relatively smaller in extent, may be removed without producing gross motor defects. Lesions involving the striatum again lead to no paralysis and simple reflexes are unimpaired. No complex patterns can be built, however, from these reflexes. For example, although the animal may be capable of performing the various activities of mating, nest building, incubation of eggs, and so on, these are performed in a haphazard fashion and are not combined in the orderly sequence that is necessary if they are to produce the proper result.

Although crude, these experiments form a basis on which it is possible to build a provisional description of the functions of the *corpus striatum* in animals where a highly developed cortex is not a complicating factor. In the first place, the striatum lowers the threshold value necessary in a stimulus before it will provoke a motor response. It can, in other words, facilitate other motor mechanisms. Secondly, it can synthesize simple reflexes into a complex pattern of behaviour. It may exert some influence over tone distribution, but it cannot on its own initiate movements, and its destruction leads to no absolute paralysis.

In mammals the position is less clear. The most obvious anatomical observation is the great structural stability of the striatum. It varies very little in those mammals that have been investigated, and what variations have been noted can be fitted into no consistent evolutionary scheme. In its connexions the same basic plan can be recognized as in the amphibia and reptiles; cortical connexions are probably present, but they are not numerous, and it is essentially a structure receiving its afferent fibres from the thalamus and discharging into the subthalamus and tegmentum.

The most detailed knowledge we have of its function is derived from clinical observation in man. Lesions of the caudate nucleus, or of the caudate

and putamen, are associated with the development of choreic and athetoid movements, while in lesions of the *globus pallidus* excessive tone, affecting mainly the larger flexor muscles, forms a large part of the clinical picture. Somewhat similar effects, the development of excessive tone and tremor, have been described in the dog following striatal lesions (Edwards and Bagg, 1923).

Although it is clearly concerned in motor function, it cannot be shown conclusively that direct and dominant control of any particular motor activity is located in the *corpus striatum*. Choreic and athetoid movements necessitate cortical activity as well as lesions in the caudate-putamen mass and integrity of the *globus pallidus*. So-called "automatic associated movements", like swinging of the arms while walking, are often absent in striatal disease, and the *corpus striatum*, and particularly the *globus pallidus*, is often claimed as the centre for such movements. I cannot do better than quote Kinnier Wilson's words in this connexion:

> This arm swinging being neither constant, essential nor purely automatic, and partaking more of the nature of movements of cooperation or auxiliary movements than of involuntary or reflex associated movements, its reduction or absence in cases of striatal disease cannot be regarded as in any way supporting a theory of the localization of "associated movements" or "automatisms" in the *corpus striatum*.

One must stress the fact that there is no actual paralysis resulting from striatal lesions. All movements are performed badly and with difficulty, so that those which are not essential, like the movements of expression or arm swinging, are cut out or reduced to a minimum.

It is particularly noticeable that it is mainly in relation to the motor functions of the cortex that the striatum is important. After decerebration in which the thalamus and subthalamus are left intact, rhythmical movements, such as walking, can be quite well performed, and the different postural reflexes are present and active. None of the features of Parkinson's syndrome appear (Dresel, 1924, and Rothmann, 1923). Choreic and athetoid movements are both at least partly cortical in origin, and in primitive mammals it has been shown that lesions involving both cortex and striatum lead to gross paralytic symptoms, which are insignificant in lesions of either structure alone. It may be relevant that in the opossum, where the *corpus striatum* is found to be electrically excitable after complete degeneration of the pyramidal system (Rogers, 1924), it produces contralateral mass extensor movements in just those parts of the body that are normally under pyramidal control.

These facts all point to some general participation of the *corpus striatum* in all cortical motor activities. This participation is in some way concerned with the control of tone; to some degree it may be antagonistic to cortical influence, since, unlike the cortex, it seems to inhibit flexor rather than extensor tone. From its stability and uniformity in all mammals one might suggest that its activity is of a primitive kind, needed equally by all mammals, and that in all it may serve very similar functions. If it works in partnership with the cortex there is no reason for assuming that it is more important in animals than in man. The relationship may be reciprocal, it is true; in an animal with little cortex the striatum may play the dominant part, while in Primates it would be subordinate to the cortex. This is commonly assumed to be the case, but it seems equally likely that as the cortex rises to importance, its partner, the *corpus striatum*, rises with it.

I doubt if in the present state of our knowledge we can get nearer to a precise definition of the functions of the *corpus striatum* in mammals than this. Some decerebration experiments (Dresel, 1924) in dogs suggest that the striatum has some capacity for combining simple movements into complex patterns, as it apparently has in birds and reptiles. How far this is a normal function of the *corpus striatum* it is impossible to say. Kinnier Wilson, in 1928, has said: ". . . the ganglia situated in the base of the brain still, to a large extent, retain the characteristics of basements—viz. darkness." His words are still true.

We are, however, in a position to sketch at least in outline the evolution of the vertebrate forebrain. The amphibia, in respect of both the cortex and the basal ganglia, are genuinely primitive. There is practically no differentiation of nuclei in either the thalamus or the *corpus striatum*. Reptiles and birds, in many ways closely related, show a striking advance so far as the basal ganglia are concerned. Individual nuclei in the thalamus have appeared, the extrinsic connexions of which are predominantly with the *corpus striatum*. The latter too has increased greatly in size, and has acquired a highly differentiated and complex structure. The cortex has remained insignificant, so that the forebrain consists essentially of a complex thalamo-striatal mechanism. This mechanism is related to a type of behaviour which, however complex it may be, is stereotyped and invariable. There is some evidence that the striatum is particularly concerned in the integration of simpler reflexes into these stereotyped patterns.

In the mammals also the thalamus has increased in size and has become differentiated into a number of nuclei. These differ in detail from those of reptiles and birds and especially in one fundamental respect. While the differentiation of the thalamus in reptiles and birds has taken place in relation to the *corpus striatum*, in mammals it has been as closely linked with the cortex. Cortex and thalamus have evolved together, while the *corpus striatum* has shown little tendency to change. It differs radically from the complex striatum of reptiles and still more from that of birds. It seems that it has evolved with comparatively slight changes from a generalized condition like that represented by modern amphibia. As the evidence of fossils shows, mammals, almost from the beginning, have followed a different evolutionary course from that of reptiles and birds, and it is very unlikely that exact homologues for the complex neo- and palæo-striatal structures of these animals will be found in mammals. There have, in fact, been two main lines of forebrain evolution, divergent almost from the beginning. The first leads to the elaboration of a complex thalamo-striatal mechanism, associated with a complex specialized and stereotyped motor behaviour, seen at its best in birds. It neglects cortical possibilities. The other leads to a thalamo-cortical mechanism, with the *corpus striatum* present, but little advanced beyond its primitive structure and function. It is associated with a type of motor behaviour which is in some ways simpler and less specialized, but which has marked plasticity and adaptability, and is seen at its best in the order Primates.

This evolutionary picture that I have attempted to draw is of necessity not clearly defined. Most of its outlines are blurred, and in many places they can be filled in only by imagination. It is open to criticism on many points, and no doubt will be altered from time to time as our knowledge

advances. Nevertheless, provided that one realizes its speculative and provisional nature, it seems to me to provide a useful background against which to examine the complexities of the human brain. Perhaps the most important impression one gains from it is that the forebrain has evolved as a whole; from the very beginning its various parts and functions are closely interconnected. Centres do not evolve in isolation, and one part of the mechanism cannot change without producing modifications throughout the whole system.

We find, for example, that it is impossible to discuss the motor mechanisms apart from the sensory. Fundamental in the whole process of forebrain evolution are the changes that have taken place in the thalamus. Through its close relationship on the one hand with the striatum and on the other with the cortex, it has been equally concerned in the development of the two main types of vertebrate forebrain. Leaving aside this fundamental sensory relationship, we have on the motor side three interrelated systems to deal with: the *corpus striatum,* the pyramidal system and the extrapyramidal cortical system. It may be of value and interest to examine at this point some of the ways in which we know these systems to be interrelated.

In man all the three systems are well developed. It is at the same time true that one, the pyramidal system, is responsible for much that is characteristic of human motor behaviour. It has risen to a position of dominance, but it does not follow that the extrapyramidal systems are superseded and no longer important. Extrapyramidal cortical areas have been shown to be capable of inhibiting extensor tone, a function that has no meaning in isolation. If this function is considered as a preparation for subsequent activity of the flexor muscles, largely under the control of the pyramidal tracts, this inhibition becomes understandable and can at least be fitted into a rational system. Tower has shown in the cat that it may also inhibit rhythmical movements like walking, a large part of the mechanism for which is situated in the subthalamus. It arrests the animal in activities of a more automatic character, and it assumes a posture of attention, a function which is again of significance as a preparation for activity of a less automatic character, probably mediated through the pyramidal tract. Other extrapyramidal areas have been shown to initiate the movements which I have described as "more automatic", and presumably a negative quiescence of the pyramidal system is as important a factor in this initiation as the positive activity of the area in question. It is obvious that we know very little yet of the precise way in which the more positive functions of the extrapyramidal systems are woven into the pattern of total motor behaviour. We are bound still to think rather vaguely in terms of facilitations and inhibitions, but a beginning has been made which may well lead to a more complete understanding of the motor functions of the cortex as a whole.

I have discussed the observations that indicate a very close relationship between striatal and cortical function. The fact that the symptomatology of striatal lesions can be profoundly affected by concomitant cortical lesions, and *vice versa,* is probably the strongest piece of evidence for this view, and it suggests that the study of such combined lesions, which do sometimes occur in man, may prove a very fruitful field of investigation. Whether it is with the pyramidal or the extra-pyramidal functions of the cortex that the striatum is most closely bound we do not know. There is evidence for some connexion

with both types of function. This general conclusion seems inevitable in spite of the fact that direct cortico-striatal connexions are few and perhaps lacking in Primates and man. It is to some extent supported by the embryological history of the cortex and *corpus striatum*. Both differentiate in close topographical relationship in the wall of the hemisphere vesicle, the cortex in its dorsal and the *corpus striatum* in its ventral part. Some observations suggest that a part of the *corpus striatum* may arise from the dorsal or cortical region of the hemisphere vesicle; certainly the two regions, cortical and striatal, are very poorly differentiated from each other in the embryo. Embryological similarities of this kind are of course by themselves no evidence for community of function in the adult structures. Taken with other evidence, however, they may be significant.

My general conclusions are meagre You may well complain that we have travelled over a great deal of ground and have picked up little, if anything, of direct clinical application. I hope, however, that I have succeeded in showing how the developmental or evolutionary approach, in combination with physiological and clinical observations, can give a more balanced and rational conception of forebrain function than could be obtained without it. It has helped to fit the thalamus in its place as the basis for all forebrain differentiation, to show how the motor cortex has crystallized out, as it were, in a general field of extrapyramidal activity, and how the *corpus striatum* has stood by, playing a primitive but essential rôle in all the motor functions of the forebrain. To attempt greater precision in a survey of this kind would, in the present state of our knowledge, be premature. We know a great deal of what happens in a single nerve fibre when an impulse passes along it, and in considerable detail. We are beginning to understand something of what takes place at the individual synapse. When we are dealing, however, with neurones *en masse*, as we always are in the central nervous system, the case is altogether different. We know almost nothing in precise detail of the way in which large numbers of neurones interact. I have suggested that the conception of more or less independent centres with defined functions is inadequate. In its place has been put a conception of complex interconnected systems, with here and there nodal points at which many paths meet, a conception which finds a ready analogy in the automatic telephone exchange. It seems to me that this is a little nearer to the truth, but I have no doubt that, in its turn, it will prove inadequate. In some respects it has already done so. Meanwhile, like all hypotheses, it will serve its purpose. We cannot investigate the precise physiology even of the pyramidal tract till we are certain exactly what cells in the spinal cord receive synaptic connexions from it, and how these cells transmit the impulse through to the anterior horn, to mention only one fundamental anatomical fact that is not yet clear. While this and many other facts are still obscure, we cannot hope to get much nearer to the understanding of the motor mechanisms of the forebrain than a crude and inaccurate analogy will take us. But at the same time, however incomplete our knowledge, it is important sometimes to pause and examine critically what has been built so far, and from other angles than that of our own particular speciality. It will often be a humiliating experience. Whether as clinicians we think of syndromes and pathological lesions, or as physiologists of centres with defined functions, or as anatomists of structure and connexions, we shall always find that the

reality is too complex to be conceived in terms of our own favourite over-simplification.

I think we should remember, too, that our methods of investigation in this particular field are not all that could be desired. The technique of physiological analysis of the central nervous system is still much the same as it was fifty or more years ago, and Pavlov's words are still apposite:

> . . . . this method naturally suffers from fundamental disadvantages, since it involves the roughest forms of mechanical interference and the crude dismembering of an organ of a most exquisite structure and function. Imagine that we have to penetrate into the activity of an incomparably simpler machine fashioned by human hands, and that for this purpose not knowing its different parts, we take a saw and cut away one or another fraction of it, hoping to obtain an exact knowledge of its mechanical working.

We do, however, make some headway, and I hope I have shown how much this is due to a close cooperation between clinician, physiologist and anatomist. If this "organ of exquisite structure and function" is to give up its secrets, it will not be to one of them, but to all these three working together.

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Surgical Technique.

THE TECHNIQUE OF VAGINAL PLASTIC REPAIR.

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Melbourne.

Preliminary Care.

WHEN first seen, many patients requiring vaginal plastic repair are tired and overwrought. If the prolapse is of the second or third degree, cervical erosion and attrition ulceration of the exposed cervix and vaginal mucosa often add to their woes. Under such circumstances it is desirable that they should be confined to bed and treated by daily vaginal packings with glycerine gauze, such application being sufficiently firm to replace mechanically the prolapsed organs. The packing is removed in the evening and a douche of "Dettol" 5% is given, after which the patient rests till the following morning, when the tampon is reintroduced.

Administration of nourishing food and regulation of the bowels can be carried out conveniently in the preliminary care, and it is remarkable how rapidly the local area improves. As a rule the patients are brought into very satisfactory condition for operative procedures in about a fortnight.

Immediate Pre-Operative Preparation.

Thirty-six hours before operation a brisk purge is administered, preferably one to which the patient is accustomed. This is repeated twenty-four hours before operation if necessary. The evening before operation a bowel lavage is given. This treatment should have been concluded at least twelve hours before the operation, in order to allow the alimentary tract to settle down.

The food taken for twenty-four hours before operation should be nutritious but of the "light" variety. On the morning of operation sweetened fluids may be given up to two hours before operation, especially if spinal anæsthesia is to be used. The only local preparation required in these cases is the shaving of the vulva. Endeavours to sterilize the perineal skin beforehand are only a source of pain to the patient and unnecessary, as preparation of the operation area is undertaken in the operating theatre after the patient has been anæsthetized.

If spinal anæsthesia is to be induced, a preliminary hypodermic injection will be given of morphine, 0·016 gramme (one-quarter of a grain), and hyoscine, 0·0006 gramme (one one-hundredth of a grain), one hour and a half before the time appointed for operation. This injection having been given, the ears are lightly plugged with cotton wool and the eyes are bandaged. The room is quietened and the patient is encouraged to sleep. No disturbance is now tolerated, and for this reason the patient is not catheterized before leaving the ward. The bladder can be emptied in the theatre after anæsthesia has been induced, while the perineal cleansing is being performed. If the patient sleeps soundly after the first injection, no further sedatives are administered. However, if she is not soundly asleep, a second injection of morphine, 0·01 gramme (one-sixth of a grain) and hyoscine, 0·00045 gramme (one one-hundred-and-fiftieth of a grain), is given half an hour before operation. The patient should be transferred to the theatre ten minutes before the time of operation.

Spinal anæsthesia is induced with two cubic centimetres of 0·5% "Percaine" (Ciba), the needle puncture being made between the fourth and fifth lumbar vertebræ. A hypodermic injection of ephedrine (Lilly), 0·05 gramme (three-quarters of a grain), should be given subcutaneously at this time to minimize any fall in blood pressure.

Local Preparation after Anæsthesia has been Induced.

The patient may be placed in the lithotomy position five minutes after the spinal injection has been made, and the skin of the vulva and perineum is mechanically cleansed with ethereal soap on gauze swabs, after which the vaginal mucosa is similarly treated. The excess soap foam is douched away with a sterile water pitcher-douche. The parts are then dried with gauze mops and 2% spirituous picric acid is painted freely over the area. The patient is draped with sterile sheets, which should be sewn to the skin of the labia in such a manner as to act as light retractors (Figure I). The anus is excluded by the suture of a gauze swab in similar fashion.

FIGURE I. Method of suturing towels to act as retractors for the labia and to exclude the perineal area.

Operative Technique.

In this description the words "left" and "right" refer to the sides of the patient.

Anterior Colporrhaphy with Amputation of the Cervix (Fothergill).

Stage I—Control of the uterus.—The operator, being comfortably seated, and provided with a suitable lithotomy table, introduces a Sim's operating speculum and exposes the cervix, which is seized and drawn towards the outlet by a tenaculum. This tenaculum is immediately replaced by introducing controlling sutures of thread into the anterior and posterior cervical lips with a large fully-curved Mayo trochar-pointed needle, the long ends of the threads being caught by short artery forceps.

The cervical canal is then sterilized with 2% picric spirit on a loaded Playfair's probe, after which a uterine sound is introduced and the length of the uterine cavity ascertained. As the aim of operation is to reduce the overall length of the uterus to approximately 7·5 centimetres (three inches), this is an important manœuvre, as it indicates how much, or how little, of the cervix must be included in the amputation. Dilatation of the cervix now proceeds until a dilatation of 11/14 Hegar is attained. The thread controlling sutures in the cervical lips are now tied together, closing the cervix temporarily, and the ends are caught by a single artery forceps.

Stage II—Incision and separation of the anterior flap, which consists of the mucous membrane and the thinned-out pubo-cervical fascia, from the underlying bladder. Mobilization of the base of the bladder from the cervix.—The cervix is now pulled downwards to the full extent of the prolapse, thus placing the anterior vaginal wall on the stretch. The main guide points are now selected and marked with light Morrison tissue forceps (Figure II). The upper central point is about half an inch behind the urethra, and it is sound practice to pick up this point with two forceps, thus flattening the angle of the incision and preventing an unsightly suburethral bulge which, if this precaution is not taken, sometimes occurs. In placing the lateral guide points, it should be remembered that when the operation is completed, they will correspond to the anterior lip of the newly formed cervix, and they should not be placed too high on the side of the cervix as it is at present exposed. When the prolapse is very marked, additional intermediate guide forceps may be placed between the main guides to assist in accuracy and control.

A triangular incision is now made joining the three main guides, which are held under light tension by the assistant while the operator deals with each side in turn.

The lateral guides are now allowed to hang loosely while the assistant draws the main central guides upwards. To raise the triangular flap two short artery forceps are applied at either edge of the top of the triangle, and by light touches with the scalpel the flap is loosened from the periurethral tissue. As progress is made, a second short artery forceps is clipped to either edge, and with all four forceps gripped in the palm of the left hand and the left index finger supporting the flap, scalpel dissection proceeds. Except in cases of very long standing, gauze dissection is not possible at this stage.

As progress continues the short artery forceps are placed more and more widely on the flap, and after about an inch of the tissue has been raised, a cellular space between the pubo-cervical fascia and the bladder is entered, and the flap may now be stripped down rapidly by gauze dissection, aided by occasional touches with the scalpel.

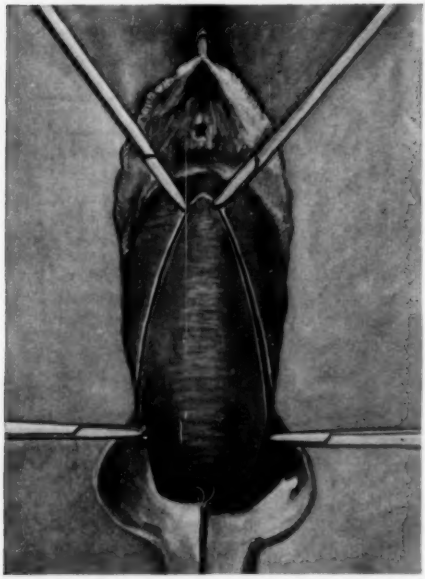

FIGURE II. The cervix has been pulled down by the thread controlling sutures, and the guide points have been selected and joined by incisions.

At this point it is wise to introduce four or five interrupted apposition sutures in the upper part of the incision to control the oozing which is at times very persistent. The suture material for apposition is number 2 chromicized catgut throughout, carried on small Mayo trochar-pointed needles. As the first suture is introduced and tied, the main central guides are removed and the suture ends are caught with artery forceps and held up so that each suture serves as a guide for the introduction of the succeeding one. When the second suture has been introduced the first one is cut away to a length of about 0·6 centimetre (one-quarter of an inch) and the second tie is clipped, cut and held up. These sutures take in mucous membrane edge and considerable pubo-cervical tissue as well, and, correctly placed, tuck the urethra backwards under the pubic arch.

The sutures are placed about 0·8 centimetre (one-third of an inch) apart; after five or six have been introduced, the final stitch is held up by its long catgut ends and suturing is temporarily suspended.

The controlling sutures in the cervix are now made taut, and, with the flap held in the left hand, separation of the bladder from the cervix in the middle line is effected by gauze-covered finger pressure. When the bladder is freely mobilized centrally it should be rolled upwards and outwards on either side by pressure with the gauze-covered thumb (see Figure III). This manœuvre carries the ureters well out of the danger zone. The bladder should be freely separated until the reflection of uterovesical peritoneum is seen. Any spurting vessels at this stage may be clipped and tied with number 0 plain catgut.

Stage III—Introduction of the "crown suture", which is the essence of the anterior colporrhaphy.—This suture picks up the base of the parametric tissues, including the insertion of the uterosacral ligaments on either side, and brings these structures forwards in front of the uterus. When the suture is correctly placed and tied, the cervix is carried upwards and backwards into the pelvis and the point of fixation of the base of the bladder is ensured.

This suture is introduced with a fully-curved Mayo taper-pointed needle carrying number 3 chromicized catgut. The needle enters the mucous membrane near the left edge of the V incision about 1·6 centimetres (two-thirds of an inch) above the lower angle guide. With the cervix pulled downwards to its greatest extent and guided by the left forefinger behind the uterus, the needle is introduced deeply outwards and backwards at the side of the uterus, and then allowed to curve forward again to

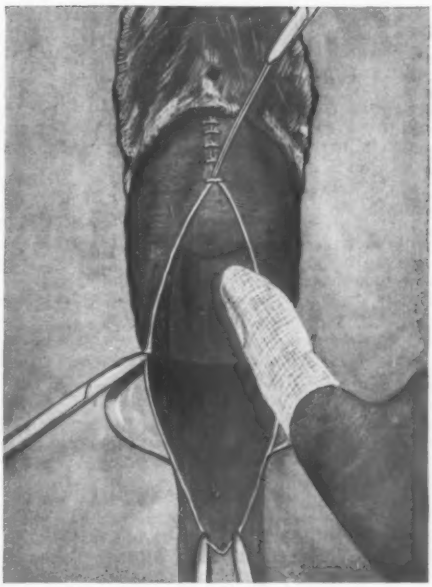

FIGURE III. The flap has been turned down; the bladder, mobilized centrally, is now being rolled upwards and outwards by pressure with the gauze-covered thumb. Certain anterior sutures have already been placed and tied.

emerge close to the side of the uterus. By means of this manœuvre the suture picks up the base of the parametric tissue and the insertion of the utero-sacral ligaments. The anterior uterine wall is then caught by this suture just below the reflection of the utero-vesical peritoneal pouch, thus determining the lowest point of new fixation for the base of the bladder. The parametric tissues on the right side are then caught by entering the needle close to the side of the uterus and allowing it to take a full curve outwards and forwards, after which the suture leaves the incised area by piercing the right edge of the V incision 1·6 centimetres (two-thirds of an inch) above the lower angle guide. The long ends of this suture are clipped to the towels which cover the patient by small artery forceps (Figure IV).

Stage IV—Amputation of the cervix and reconstruction of the portio vaginalis by the insertion of sutures of the Sturmdorff type.—Amputation of the cervix is now to be undertaken, and as the aim of this procedure is to reduce the overall length of the uterus 7·5 centimetres (three inches), the amount of tissue to be removed depends on one's operative findings. This step may occasionally be omitted, particularly if the uterus is atrophic and the cervix appears to be quite healthy. The controlling sutures in the cervix are drawn firmly upwards by the assistant, exposing the back of the cervix, and an incision is made joining the two lower angle guides, curving round the back of the cervix in its course. A flap of the posterior vaginal wall is then rapidly raised all along the line of this incision to a depth of about 1·25 centimetres (half an inch). A brisk ooze may occur at this stage and is not controlled till the Sturmdorff inverting sutures are placed, so that dexterity at this stage considerably lessens the amount of blood loss.

The cervix has been completely circumcised, and the tissues at the sides of the cervix, carrying the descending cervical branches of the uterine artery, are caught with ibis forceps and severed with the scalpel down to the actual cervical tissue. These pedicles are immediately undersewn and tied off with number 1 plain catgut on half-circle Mayo needles.

With tension once more on the controlling sutures, the cervix is amputated cleanly, and the cervical

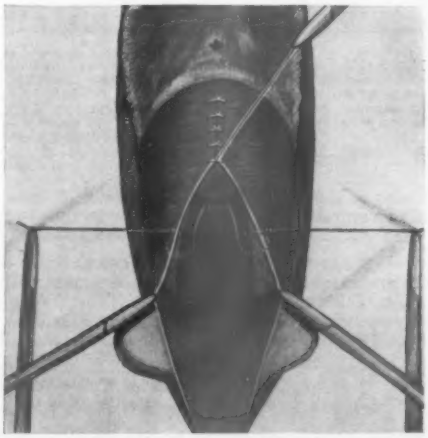

FIGURE IV. The "crown suture". The deep sweep of the suture on either side, picking up the bases of the parametric tissue, is shown by the dotted line.

stump is seized with tenaculum forceps by the assistant, while the operator immediately proceeds with the introduction of the inverting sutures which will reconstitute the cervix. These sutures are of number 2 chromicized catgut, carried on fully-curved Mayo trochar-pointed needles. The mid-point of the posterior flap is picked up with the loop of the suture inside the flap, and the needle is then entered at the junction of the cervical canal and cervical tissues and passed downwards and backwards, following the curve of the needle, till it emerges about 2·5 centimetres (one inch) behind the mid-point of the flap. The needle is then unthreaded, and, the other end of the suture having been threaded in, it is introduced at the same point and passed through the posterior cervical tissues, emerging about 0·3 centimetre (one-eighth of an inch) from its fellow (Figure V). This Sturmdorff suture is immediately tied and cut, and in a large measure controls the oozing from the tissues behind the cervix.

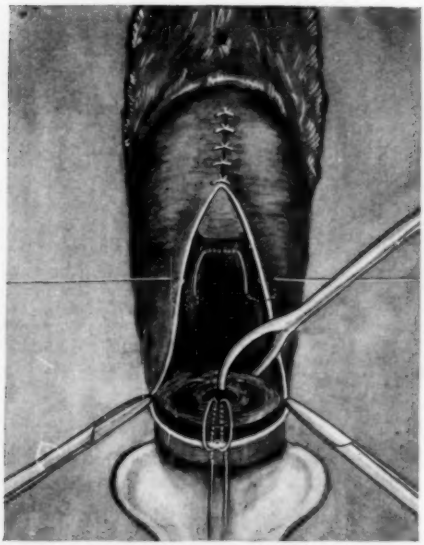

FIGURE V. The cervix has been drawn upwards and circumcised posteriorly, and the posterior mucous membrane flap, which will reconstitute the cervix, has been raised. The cervix has been cleanly amputated and the first Sturmdorff inverting suture is in position prior to being tied. For diagrammatic clarity, the crown suture is placed rather higher than in actual practice.

Two further similar sutures are now placed, taking the mucous membrane flap just below mid-centre (at about the 4 o'clock and 8 o'clock positions) and drawing it to the lateral portions of the amputated cervix.

The anterior portion of the cervix is reconstituted by a Sturmdorff suture which is introduced "running", and not, as heretofore, by unthreading the needle. This stitch enters the vaginal mucous membrane 1·25 centimetres (half an inch) from the lower angle guide on the left, and is then carried vertically through the anterior cervical tissues to emerge at the edge of the cervical canal, after which the left angle of the vaginal flap is secured from within outwards, the guide being removed to permit of this. The angle guide on the right side is then removed and the suture enters this vaginal flap angle from without inwards, then traverses the anterior cervical tissues in an upward direction, finally leaving the incision through the vaginal mucous membrane on the right side from within outwards (Figure VI). When this suture is tied, the cervix is completely restored and all hæmorrhage is controlled.

Stage V—Tying of the "crown suture" inside the vagina.—The "crown suture" should now be tied, with the fingers pressing the cervix upwards and backwards into the pelvis as this is done. It is then cut, the ends being left about 1·25 centimetres (half an inch) long.

Stage VI—Closure of the remainder of the wound.—The remainder of the V incision, now straight, is apposed by interrupted number 2 chromicized catgut stitches inserted from behind forwards, the effect of each succeeding suture being to lift the cervix further back into the pelvis. The field is now quite dry and the vagina should be swabbed free of blood clot before the posterior work is commenced.

Posterior Colpo-Perineorrhaphy (Donald).

Stage I.—The basis of this work is the excision of a long triangular flap from the posterior vaginal wall and the isolation and reconstruction of the separated levatores ani muscles. As this flap is in many cases six or seven inches in length, it is unwieldy to handle, and for the purposes of accuracy and control the excision is performed in stages, a suitable triangular area being marked out, raised and at once sutured in a vertical direction at each stage.

With the aid of the Sims speculum, the highest point of the rectocele is selected and picked up with light Morrison tissue forceps, and two lateral guide points in the vaginal sulci are chosen so that an equilateral triangle is marked out. The guide points are joined by suitable incisions and the triangular flap is raised by gauze-covered finger pressure. Excessive oozing at this stage is usually caused by the tissues being separated at too great a depth. Usually an avascular plane can be found. This incision is immediately closed in a vertical direction by interrupted sutures of number 2 chromicized catgut in a small Mayo trochar-pointed needle (the clip, cut and hold up principle—*vide supra*). As each stitch is tied, the preceding tie passes further up the vagina. When the two lateral guide points have been approximated, the tie is made and the long ends are held up while the Morrison forceps are moved.

Stage II.—The last suture tied and held up "long" marks the apex of the next triangle to be marked out and dealt with.

Lateral guide points are selected and, at this stage, the usual points are the remnants of the last hymenal caruncle on each side (see Figure VII). From the apex of this triangle incisions are made to the lateral guides and the flap is raised further, the procedure being now easier as the plane of separation has been recognized in the first triangle.

The anterior rectal wall becomes visible in the depth of this incision, and if unduly redundant may be plicated in a vertical direction. This step, however, is not part of the standard technique.

The suturing of the edges of the incision in a vertical direction is then continued, a good grip of the mucous membrane and underlying tissues being taken in each stitch until the hymenal caruncles are almost approximated, when again the last tied stitch is held up "long" by the assistant.

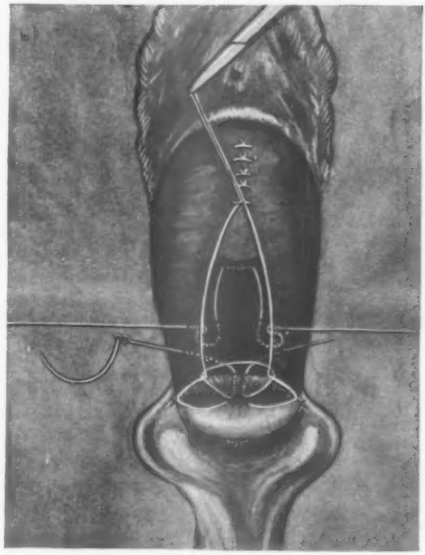

FIGURE VI. Three Sturmdorff sutures have been placed and tied. The course of the "running" Sturmdorff suture which finally reconstitutes the cervix is shown.

Stage III.—This suture now marks the apex of yet another triangle to be excised. Guide points on the muco-cutaneous junction are now selected and picked up with light Morrison forceps; their approximation at this time should allow easy three-finger introduction, as the introitus will be considerably narrowed by further suturing. At this junction the thread guides holding the labia to the covering sheet should be cut and the labia released. This flap is marked out with the scalpel and rapidly raised, usually by scalpel dissection, as there is often a great deal of scarred tissue in this area.

Stage IV—Identification and reconstruction of the levatores ani muscles.—The anterior rectal wall is firmly pressed backwards with the gauze-covered finger and the separated edges of the *levatores ani* muscles are now distinctly apparent in each lateral sulcus. These may be seized with light tenacula (towel forceps serve admirably) and demonstrated clearly in the depths of the wound.

These muscles are approximated by a suture of number 3 plain catgut carried in a large fully-curved Mayo taper-pointed needle, which is introduced deeply and carried widely round both muscle fasciculi, then picking up the fascia-covered edges of both muscles close to the middle line, thus making a "near and far" approximation (Figure VIII). This suture is tied firmly, but not so tightly as to strangulate the muscle, a frequent error which may lead to the breaking down of the perineum later on. Two further sutures are now introduced to continue the approximation of the *levatores ani* down towards the *sphincter ani*. These sutures are all cut very close to the knot.

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS.
GORDON CRAIG LIBRARY.

Stage V—Binding of the newly formed posterior vaginal wall to the levatores ani muscles.—When the *levatores ani* have been neatly apposed, the operator returns to the apex of the mucosal flap, which is clearly defined as the assistant exerts traction on the long holding suture. A mattress type suture of number 2 chomicized catgut is now introduced, entering the mucous membrane near the last caruncle on the left side, and passing through the top of the approximated *levatores ani* and out through the mucous membrane on the other side. When tied, this suture firmly binds the newly formed posterior vaginal wall down to the *levatores ani* and effectually controls what has often been a rather persistent oozing from small vessels in this area. Each suture introduced from now on is cut immediately it has been tied.

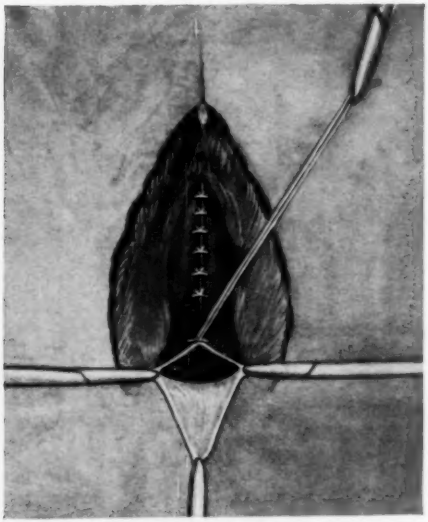

FIGURE VII. The first triangle of the posterior colpo-perineorrhaphy has been excised and sutured and the final stitch held up "long". Guide points on the last hymenal caruncles have been selected, after which the excision of the second triangle proceeds by incision of the mucous membrane from the apex to each lateral guide and dissection.

Stage VI—Fashioning of the perineal flap and restoration of the vulval and perineal tissues.—The anal occlusive swab is now removed by cutting the thread, and the fashioning of the new perineal flap now proceeds. The perineal skin is picked up in the middle line about 1·87 centimetres (three-quarters of an inch) in front of the anus with Morrison forceps, and a triangular flap with its base upwards is marked by joining the two lateral guides on the muco-cutaneous junction to this central point (Figure VIII). This flap has usually to be excised by scalpel dissection, after which the lateral tissues representing the remains of the urogenital diaphragm are approximated with number 1 plain catgut interrupted sutures, the ends being cut very close to the knot in order to leave as little extraneous matter in this area as is possible. Two layers of sutures can usually be conveniently introduced here, the aim being to avoid leaving any dead spaces in the newly formed perineal body. Any mucous membrane edges requiring apposition can now be brought together with number 1 chromicized catgut, and the perineal skin edges are finally apposed by three Michel clips. The whole aim of perineorrhaphy should be junction without tension, as strangulation of the tissues is the most potent cause of breaking down of the operation area in the later stages of convalescence.

The new vagina, which now admits one finger comfortably, is dried out with three small gauze mops, the first moist, the second dry and the third dipped in 2% picric spirit. Vaginal packing is neither desirable nor necessary. The perineal area is swabbed with picric spirit also and a sterile pad applied over the vulva. This is maintained in position by means of a hand towel, folded in three and attached in front and behind to an abdominal binder, an arrangement having great advantages over the usual T-bandage, which can be extremely uncomfortable.

Post-Operative Care.

If a spinal anæsthetic has been used, the patient may have a warm drink on return from the theatre. She is placed flat in bed with one small pillow, the foot of the bed being raised on twenty-five-centimetre (ten-inch) blocks for eight hours, or until such time as she can move both feet freely. It must be remembered that the patient is quite insensitive from the waist downwards and great care must be taken with hot water bottles. After the effects of anæsthesia have passed off, the foot of the bed is lowered and the patient is allowed to assume a comfortable reclining position with sufficient pillows for comfort.

Diet.—Vomiting is not a marked feature, and the patient may have a light diet, with barley sugar and glucose in drinks. It is rarely necessary to subject her to the rigours of a "non-residual diet". The taking of fluid is encouraged, and the patient assured that she will suffer no discomfort if she has retention.

Urine.—Retention of urine is the rule in these patients, in view of the extensive separation of the base of the bladder from the surrounding structures.

The patient will be catheterized eight-hourly (or more frequently if uncomfortable) until such time as she can void, after which a catheter should be passed once in twenty-four hours, immediately after voiding, until such time as there is no residual urine. If management of the bladder on these lines is practised, post-operative cystitis (in practically every case due to presence of residual urine in an atonic bladder) will be eliminated. In any case, the use of "Hexostate" tablets in the post-operative period is to be commended.

Bowels.—The bowels are usually kept closed for five days after operation. It is sound practice to give the patient fifteen cubic centimetres (half an ounce) of plain liquid paraffin night and morning from the third morning onwards. Castor oil is the most suitable purgative, and thirty cubic centimetres (one fluid ounce) are administered on the fifth evening. In most cases an easy bowel action follows. The use of paraffin usually avoids the necessity of giving a maize oil enema just before the bowels act, a procedure which is not devoid of risk.

Sedatives.—Provided that there is no morphine idiosyncrasy, hypodermic injections of 0·01 gramme (one-sixth of grain) of morphine give these patients adequate rest and lessen the tendency to unwanted bowel activity. Once the bowels have been opened these patients have very little discomfort and simple sedatives are all that are required.

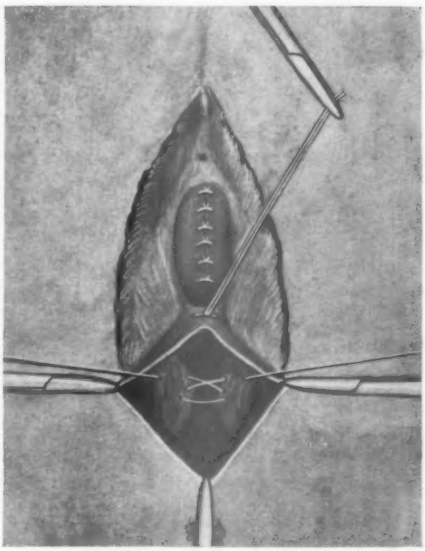

FIGURE VIII. The triangle seen in Figure VII has been excised and the wound sutured, the final stitch being held up "long". Guide points are seen on the mucocutaneous junction; the central perineal guide is in position and both mucous membrane and cutaneous triangular flaps have been excised. The *levatores ani* muscles are exposed in the depths of the wound and a coaptation suture (the middle one for clarity) is seen in position before being tied.

Perineal Toilet.—In probably no one surgical procedure is success more dependent upon good nursing after-care than in this operation. It is essential that the perineal area be kept dry in the post-operative period and, with this aim in view, the perineal pad must be changed frequently, even hourly at first, and a routine toilet carried out. After the first forty-eight hours, this toilet is carried out four-hourly and also at any time the bladder or bowels are evacuated: The patient is placed in the left lateral position, and, the soiled pad being removed, the perineum is mopped dry with wool swabs, after which the suture line is lightly touched with small swabs dipped in spirit. The perineal area is then dusted with an astringent powder, such as bismuth formic iodide or bismuth subgallate, and a sterile pad again applied.

Skin Clips.—The skin clips are removed the day after the bowels are opened, and union by first intention is to be anticipated if the perineum has been kept dry successfully.

Convalescence.—Patients are usually kept in bed for sixteen days and, after cautious mobilization, usually go home in three weeks from the time of operation.

Case Reports.

TWO CASES OF DIAPHRAGMATIC HERNIA.

By H. G. Humphries,
Sydney.

The two cases of diaphragmatic hernia to be described present points of unusual interest. In both most of the abdominal viscera were found to occupy the left thoracic cavity, but in neither, after careful radiological examination, both direct and with opaque media, was it possible to determine with any degree of certainty the condition

Figure I.—Case I. Skiagram following opaque meal. Stomach occupying low level.

Figure II.—Case I. Skiagram five hours after opaque meal. Meal passing directly from stomach into small intestine occupying position in thorax.

of the affected half of the diaphragm. Although the radiographic appearances were very similar, the conditions as found at operation were very dissimilar. In the first case there was a circular opening in the left dome of the diaphragm, while in the second the almost complete lack of development of the muscular elements of the left half of the diaphragm resulted in a form of eventration for which no surgical treatment would appear to be of any avail.

CASE I.—The patient, a male, aged two and a half years, was admitted to hospital with a history of attacks of vomiting, occurring every week or so, for the previous four months, the attacks becoming more frequent and accompanied by severe pain.

At operation in February, 1937, I resected about 12·5 centimetres (five inches) of the left eighth rib, and on opening into the pleural cavity I found a very extensive diaphragmatic hernia, lying uncovered by any sac, in the left side of the thorax, and consisting of most of the small intestine, the large bowel as far as the splenic flexure and the spleen. The collapsed lung occupied a small space above the level of the third

FIGURE III.—Case I. Skiagram showing opaque enema passing directly from splenic flexture into large bowel occupying position in thorax.

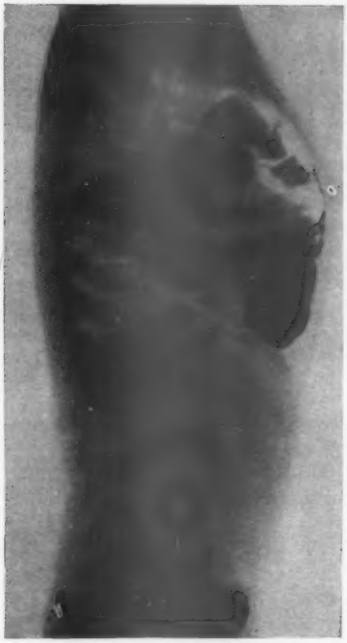

FIGURE IV.—Case I. Skiagram following opaque enema. Lateral view.

rib, the heart being pushed over to the right of the sternum. There was a circular opening in the diaphragm about 3·75 centimetres (one and a half inches) in diameter slightly postero-lateral to the summit. An abdominal incision parallel to the left costal margin was made, and with considerable difficulty the contents were reduced through the opening; but as they had been so long resident in the thorax, the abdomen had remained undeveloped and was unable to accommodate the additional mass. Any further attempt at surgical treatment therefore was abandoned.

The child's condition was very precarious during the operation; he did not recover from the shock experienced and died on the following day.

CASE II.—The patient, a male, aged four and a half years, was admitted to hospital in March, 1938. A few months after this boy's birth his mother had observed the pulsations of the heart on the right side. There was no history suggestive of partial intestinal obstruction as in Case I.

Skiagrams showed the left lung to occupy a small space above the level of the fourth rib, with the stomach and splenic flexure of the large bowel in close apposition.

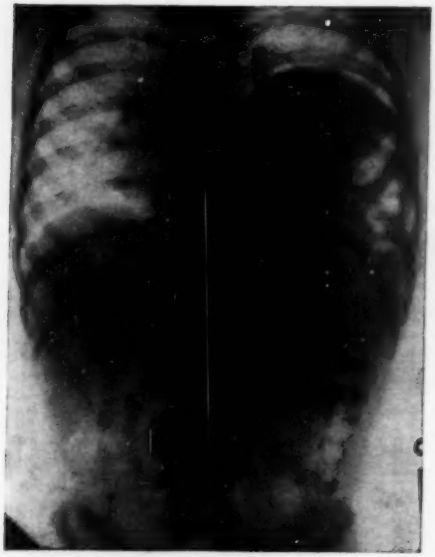

FIGURE V.

FIGURE VI.

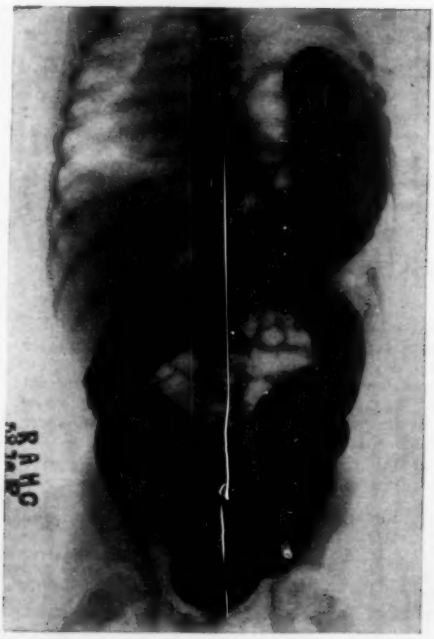

FIGURE VII.

FIGURE V.—Case II. Skiagram following opaque meal. Stomach enormously dilated and folded on itself.

FIGURE VI.—Case II. Skiagram showing stomach and splenic flexure with some coils of small bowel occupying position in left side of thorax.

FIGURE VII.—Case II. Skiagram following opaque enema demonstrating the high level of the splenic flexure.

At operation on March 18, 1938, by combined abdominal and thoracic routes, I was disappointed to find an extensive eventration, the displaced abdominal contents consisting of a very dilated and thickened stomach folded on itself, some coils of small intestine, dilated and hypertrophied coils of large bowel in the region of the splenic flexure, the spleen and portion of the left lobe of the liver. The left half of the diaphragm was represented by a narrow ring of muscle about 1·25 centimetres (half an inch) in width, along its costal margin, the remainder consisting of a ballooned flaccid pouch covered with pleura and lined with peritoneum containing the viscera mentioned.

Nothing further was attempted. The child manifested no ill-effects from the laparotomy and made an uninterrupted recovery.

A CASE OF INTESTINAL OBSTRUCTION, WITH EXTENSIVE SMALL BOWEL RESECTION.

By Archie Aspinall,
Sydney.

The patient, a male, aged fifty-one years, a labourer, was admitted to a medical ward at Sydney Hospital at 2.30 p.m. on November 27, 1937, with a tentative diagnosis of coronary occlusion.

The history was to the effect that earlier in the day, at 7.30 a.m., he was in a tram, feeling quite well, when he was seized with severe pain in the epigastrium. He had no dyspnœa, but nausea and sweating were present. On admission to hospital he localized the pain in the left hypochondrium.

There was no history of any previous illness or operation. His bowels had not been opened for two days, but this was not unusual.

On examination he was pale and restless. There was rigidity of both *rectus abdominis* muscles, most marked in the hypogastrium. He exhibited tenderness in the left hypochondrium and in the lower abdomen. There was slight distension below the umbilicus. His pulse rate was 46 and his temperature 36·7° C. (98° F.). There were no abnormal findings on rectal examination. A milk and molasses enema and pituitrin were given without result. He vomited once just prior to operation.

Operation.

Operation was carried out at 9.20 p.m. the same day, a tentative diagnosis of mesenteric thrombosis having been made. The abdomen was opened through a right paramedian incision. Free blood was present in the peritoneal cavity and had a putrid odour. Dark red gangrenous small intestine was found. Several feet of ileum were found protruding through a loop of omentum. Oxygen was given and the bowel freed, but its colour did not return. The vessels in the mesentery were constricted, and the bowel was twisted on itself. A section of small bowel 2·7 metres (nine feet) in length was resected, and a lateral anastomosis was performed. About 30 centimetres (twelve inches) above the anastomosis a small tube was inserted into the proximal end of the bowel, passed through the omentum and brought out through a stab wound.

Progress.

On return to the ward the patient was put in a Fowler bed and given "Carbogen" liberally, and received a continuous infusion of 10% glucose in normal saline solution, seven and a half litres in all being administered in three days. On the third day he vomited a little and the saline was discontinued. Water only was then given for twenty-four hours. After this he was given whey, albumen water and milk hourly in small quantities. His bowels were opened forty-eight hours after operation, and he had a slight tendency to diarrhœa.

On December 1, 1937, the ileostomy wound was discharging freely.

On December 6 the use of the Fowler bed was discontinued, and the patient was given light diet.

On December 7 there was a small discharging area in the centre of the wound. The ileostomy wound was still discharging.

On December 18 the ileostomy had not quite healed.

On December 20 the ileostomy wound had closed. There was a tendency to slight constipation.

On December 29 radiographic examination was carried out. No organic lesion of stomach or duodenum was found.

On January 7, 1938, the patient was discharged from hospital.

In March, 1938, he reported that, except for slight pain in the region of the stab wound, he was in excellent health, he had put on 12·6 kilograms (two stone) in weight, and his bowels acted normally. He reported back for observation in October, 1938, at which time he had maintained this weight and looked well.

Comment.

The case is of interest on account of the absence of any history of bowel trouble, the site of the pain and the initial difficulty of diagnosis, also the early return of normal bowel function and increase in body weight.

The following notes, dealing with extensive resection of the small intestine, are of interest:

"Treves measured the small intestine in 100 bodies, and found the average length in the adult male to be 22 feet 5 inches (683 centimeters), and 23 feet 4 inches (706 centimeters) for the adult female. The shortest intestine was 15 feet 6 inches (471 centimeters), the longest 31 feet 6 inches (960 centimeters)—a difference of more than 16 feet (489 centimeters).

"The most extensive resection of small intestine recorded to date was performed by Jerrauld, who excised 19 feet (579 centimeters) of small intestine for gangrene caused by an embolus of the superior mesenteric artery. The patient was subsequently operated upon by Washburn for intestinal obstruction due to adhesions, at which time 10 feet (305 centimeters) of small intestine remained. In the joint publication made by the two authors, the patient was reported to complain of periodic spells of diarrhea, but at the last examination three years after the first operation the patient weighed five pounds more than before the initial operation.

"A patient operated upon by Doerfler appears to have survived on a remaining portion of small intestine so short that Doerfler is prompted to ask whether man may live without the small intestine. He resected 18 feet 8 inches (560 centimeters) of small intestine for a volvulus of the entire small bowel of thirty hours' standing. Twelve centimeters remained below the duodeno-jejunal juncture and 20 centimeters above the cecum. Six and a half years later, when Deorfler made his report, the patient was in good health and partook of food only at regular mealtime and ate in ordinary amounts; his bowels moved normally twice a day. For some time immediately following the operation, however, the patient was distressed by bowel movements every four hours and was considerably disturbed by gas; he had then found it necessary to eat something every four hours.

"Denk has reported an instance in which Brenner excised 18 feet (540 centimeters) of small intestine in the operative cure of a strangulated hernia. Two and a half months, as well as a year and a half after operation, absorption experiments demonstrated a mild disturbance in the absorption of fat. The patient maintained his weight of 48 kilos., however, and only complained of a moderate diarrhea. Shortly after the second observation, the patient appeared to decline and lose in strength and weight and developed real hunger despite the liberal ingestion of food. Death occurred two and a half years after operation. At that time the weight was only 26·5 kilos. At *postmortem* 3 feet 5 inches (106 centimeters) of small intestine remained."[1]

Reference.

[1] Owen H. Wangensteen: "The Therapeutic Problem in Bowel Obstructions."

A CASE OF OSTEOMYELITIS OF THE FRONTAL BONE COMPLICATING LEFT-SIDED FRONTAL SINUSITIS.

By H. H. Harrison,
Sydney.

The patient, a male, aged sixteen years, was admitted to hospital on December 29, 1937. He gave a history of having suffered from a slight head cold, and on December 24 he noticed some headache. The pain recurred the following day and became worse

Figure I. Photograph of patient showing final result.

on each succeeding day. Swelling of the left frontal region was first noticed on December 27, and became more marked on succeeding days. At no time was any great degree of nasal discharge noticed.

On examination the patient was very hot and drowsy and looked very ill. A diffuse swelling was present over the frontal region, more marked on the left side, where it extended practically to the hair-line. Some swelling of the eyelids was present, but no displacement of either eye was detected. Marked tenderness was present over the region of the left frontal sinus, extending upwards and to the left. Examination of

the nose showed septal deflection to the left, high up. The mucous membrane was congested. No pus was seen in either nostril. Nothing abnormal was detected in the post-nasal space.

Under intratracheal ether and "Carbogen" anæsthesia, an incision was made through the left eyebrow and down the left side of the nose. Pus was found under the pericranium. The bone was red and pitted in appearance. At the first chisel-cut into the frontal sinus thick pus escaped under great pressure. The anterior wall of the sinus was removed, and it was found that the frontal bone above the sinus was badly infected. As the patient was quite young, it was decided to attempt to minimize subsequent deformity by chiselling away the bone carefully, leaving the inner layer intact. This was done until healthy bone was encountered, about 2·5 centimetres (one inch) above the upper margin of the sinus. The ethmoid cells were thoroughly exenterated, a large opening was made at the site of the fronto-nasal duct, and a large tube was inserted and sutured to the nostril. The external wound was left open and packed with iodoform gauze. The condition of the patient after operation was poor.

On December 31, 1937, the patient still looked ill and was extremely tender over the right frontal sinus. There was extension of the swelling further beyond the hair-line.

Under intratracheal ether and "Carbogen" anæsthesia, operation was again carried out. The right frontal sinus was opened, and both wounds were joined by an incision across the root of the nose. A large flap of frontal tissues was lifted up, and the right frontal sinus was found to be very small and to contain no pus. The bony partition between the two sinuses was infected. This was removed, and a right radical frontal sinus operation was performed. The swelling and œdema were explored on the left side. Further patches of osteomyelitic bone were chiselled away. The wound was left open and packed with iodoform gauze.

In addition to the usual post-operative treatment, five cubic centimetres of "Prontosil" were given intramuscularly each day, and two tablets of "Prontosil" orally three times a day.

Improvement gradually took place in the patient's condition; the temperature fell, and about three weeks later it was possible to resuture the flaps into good position.

The patient's condition is now good and he is symptom-free. There is very little deformity.

The Australian and New Zealand Journal of Surgery.

All articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without any abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given, with full date in each instance.

When illustrations are required, good photographic prints on glossy gaslight paper should be submitted. Line drawings, charts, graphs and so forth should be drawn on thick white paper in indian ink. Authors who are not accustomed to prepare drawings of this kind, are invited to seek the advice of the Editor if they are in any doubt as to the correct procedure. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available.

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THE CLOSURE OF SURGICAL WOUNDS.

RECENT observations in the behaviour of various healing wounds, which have been produced and treated by different surgeons, have shown that there are far too many mild infections, and too many cases of thickening and inflammatory reaction in the depths, which result in delayed healing and a state of affairs which is far from ideal. Reactionless primary healing must be the aim and should be attained in practically all clean operation wounds. While the results in a certain number of the less satisfactory cases are due to actual bacterial contamination, whether from the skin of the patient or from slight error of technique or from inefficient sterilization, it appears that in many cases there is another explanation. It is true that recent investigations in various parts of the world have shown that catgut is not so easily sterilized as we once believed, that the efficacy of heat sterilization by the commonly used steam sterilizer is far from perfect, and that lotions and even alcohol may often harbour low-grade, but rather resistant, organisms or spores. In spite of this, however, more thought and care should be given to the detailed technique of wound closure than is often done. Anyone who has watched surgeons in many parts of the world carrying out this simple procedure, cannot help being struck not only by the diversity of methods used, but even more by the variability of the healing process. Taking for

granted the efficacy of sterilization, and in the main this is justifiable, the most important thing to bear in mind is that suture material only serves as a splint for the time being, and that the real mechanism of healing is the formation by natural processes of a thin scar of fibrotic tissue, which ideally should be as narrow as possible and which should restore the tissues to their anatomical normal. Excessive reaction in a wound must follow the use of too much suture material. Yet how often do we see surgeons using numerous layers of catgut in a clean wound much in the same way as a seamstress darning a defect in a fabric. All of this catgut has to be removed by leucocytes, and if one examines sections of wounds with their catgut embedded taken after three or four days, the amount of cellular reaction practically amounts to an abscess, aseptic as a rule, it is true, but nevertheless the generator of an excessive amount of new fibrous tissue. It is doubtful, for example, whether the commonly used subcutaneous fat suture, so often used to obliterate dead space, is better than a firm dressing, and it is almost certain that strangulation of fat by a non-removable continuous suture of this type is one of the commonest causes of induration and thickening in the subcutaneous portion of many clean wounds. I am personally convinced that the majority of surgeons use far too much suture material in ordinary clean wounds, and that, other things being equal, the less suture material used, the less reaction and the more perfect primary union do we get. The nature of the suture material used is undoubtedly important in any consideration of the mode or rate of healing, and in spite of much investigation we still lack the perfect universal suture material.

Catgut has for a long time and in many countries been very commonly used as a suture material for all sorts of tissues. It is true that it is ultimately absorbed and that it is, therefore, relatively safe in cases in which asepsis is under suspicion; but it does cause a great deal of leucocytic reaction, which is at the same time the mechanism of its absorption. During the natural removal of catgut a gelatinous substance is formed by liquefaction, and this not only is an excellent culture medium, but also engenders excessive tissue reaction. Catgut is thus much more irritating to the tissues than silkworm gut or silk, and a surgeon who has had to reopen an operation wound after a few days cannot help being struck by the amount of cellular exudate present. Provided that asepsis can be guaranteed, and this should be possible in most clean cases, there can be no doubt that fine silk is a much better suture material and that it will give rise to much more perfect wound union. This has been particularly exemplified by the work of Halstead and his pupils, particularly by Cushing, who regularly obtained the most perfect reactionless healing in scalp wounds by the use of fine, easily sterilized silk. Neurosurgeons, who use this material a great deal, are convinced of its superiority if one desires perfect reactionless primary union. I would suggest

in clean surgical wounds an extended trial of fine silk, which has the advantages that it can be sterilized properly, that it can be cut at the knot, that it is non-irritating to the tissues, that it does not slip, and that a minimum quantity of foreign material is left in the wound. I have used it for many years for the deeper parts of operations on the neck, in dissection of glands, in operations for hernia and in other clean operations in the extremities, and have never regretted it.

There can be little doubt that continuous sutures are quicker to insert and at the time of operation give in most cases a prettier and more satisfactory closure, but it is doubtful whether this condition is still present after a few days. No matter how continuous sutures are used, and they are usually pulled too tight, they are prone to produce strangulation of some tissue between the loops, and so interfere with blood supply, nutrition and healing. They are relatively traumatic, and are thus liable to lower the vitality of the tissues and so lay the foundation of low-grade infection, and if one loop does happen to slip or break, the security of the whole suture line is imperilled. Except where very accurate hæmostatic or watertight closure of thin layers, such as the peritoneum or bowel, is necessary, the use of continuous sutures should be very limited and simple—in fact, the simpler, the better. Non-strangulating interrupted sutures will be found the best means of inducing reactionless clean healing.

It is true that many other factors, such as trauma to the tissues by rough handling, the presence of small hæmatomata, the degree of adiposity of the subcutaneous tissue and the patient's personal "tissue" characteristics, are also important in causing interference with the process of normal primary union. I would, however, like to emphasize the fundamental factors mentioned, and suggest that surgeons should not rest content with the present state of affairs, and that the use of as little suture material as possible, the use of silk in an increasing number of clean cases, and the use of interrupted in preference to continuous sutures, will do much to give that perfect union that our patients and ourselves have every right to expect in clean operation wounds.

HAROLD R. DEW.

Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

The Treatment of Fractured Os Calcis by Westhues's Method.

W. Ehalt: *"Dasmodifizierte Verfahren nach Westhues zur Behandlung der Fersenbeinbrüche"*, *Der Chirurg*, September 1, 1937, page 651, and *Zentralblatt für Chirurgie*, Number 3, 1937, page 159.

H. Westhues: *"Eine neue Behandlungsmethode der Calcaneusfrakturen"*, *Zentralblatt für Chirurgie*, Number 17, 1935, page 995.

H. Bürkle de la Camp: *"Zur Fersenbeinbruchbehandlung"*, *Zentralblatt für Chirurgie*, Number 16, November 16, 1936, page 985.

In 1933 Westhues described a new method of treatment of fracture of the *os calcis*, in which he restored the axis of the *tuber calcanei* by inserting a nail through the back of the heel just below the insertion of the *tendo Achillis*. The nail was then used as a lever and plantar-flexed together with the foot (Figure II). Bürkle de la Camp used

Figure I.

Figure II.

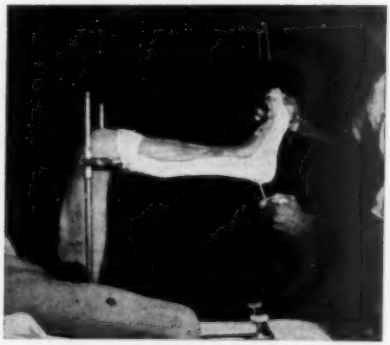


Figure III.

instead of the nail a long screw and then used traction on the heel after the above manœuvres. Neither of these surgeons paid much attention to the broadening of the heel. W. Ehalt, assistant at Lorenz Böhler's hospital, has used the method in twelve cases, and his technique is as follows. The fractured limb is elevated for five to eight days until swelling subsides. Under spinal analgesia the Steinmann nail is driven into the long axis of the *os calcis* below the insertion of the *tendo Achillis*, but below the fracture site. Westhues places lead markers on the skin, and after radiographic examination these indicate the direction and the depth to which the nail should be inserted. Now by simultaneously plantar-flexing the nail and forefoot, both the tuber angle and the arch of the foot are restored. If the lateral skiagram shows good correction, then the Böhler compressing clamp is used to correct the broadening of the *os calcis*. The plaster cast is applied whilst the assistant holds the heel in plantar-flexion and adduction by the transfixing nail (Figure III), and the forefoot in pronation with dorsiflexion of the toes. After four to five days the patient is allowed to walk, and the nail is removed in five weeks. A new walking cast is fitted

for a further three weeks. Westhues left the nail in the cast for four weeks, and then only after removal of the nail allowed walking in a cast for two to three weeks. Ehalt, however, considers that this period of immobilization is too short, that redisplacements may occur, and that the method is only suitable for fractures of Group IV and less severe forms of Group V (Böhler). Figure V illustrates

FIGURE IV.

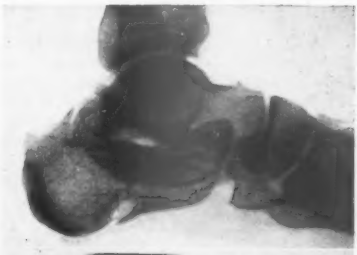


FIGURE V.

FIGURE VI.

FIGURE VII.

a severe form of Group V fracture, in which there are flattening and loss of the angle between the upper surfaces, and also an upward displacement of the posterior fragment. Figure VI shows that the upward tilting of the posterior fragment has been corrected by Westhues's method, but not the upward displacement of the fragment as a whole. Figure VII shows how, with the pin through the *os calcis* from side to side, and then with traction of the screw traction apparatus, the upward displacement has been corrected. Westhues's method is a simple one, but can only correct tilting of the posterior fragment in the long axis of the calcaneus, not the upward or parallel displacement. It has its place in the treatment of the fractured *os calcis*. Figure IV shows the walking cast with a pin incorporated. The reviewer has found that weight bearing in such a cast predisposes to sinus formation by the pin, and that in any case weight bearing under three to six months, even in a cast, can cause collapse of the *os calcis* and favours the development of traumatic osteoarthritis in the subastragaloid and other joints.

THOMAS KING.

PRINCIPLES IN THE TREATMENT OF FRACTURES OF THE CLAVICLE AND SPINAL COLUMN.

L. Böhler: *"Grundsatzliches zur Behandlung von Schlusselbeinbruchen und Wirbelbruchen"*, *Monatsschrift für Unfallheilkunde und Versicherungsmedizin*, Number 8, 1936, page 387.

LORENZ BÖHLER formerly treated all recent clavicular fractures without bandages and with active movements from the first day, whilst Magnus, according to his book, "Fractures and Dislocations", has for severe displacements placed the patient in bed and applied traction to the limb on the affected side. The traction employed since 1933

has been strapping or even a Kirschner wire passed through the olecranon. Böhler, after observing in old soldiers the pain caused in malunited fractures of the clavicle by the skin pressure from the shouldered rifle, devised in 1918 his clavicle splint, which aims at obtaining a cosmetic in addition to a good functional end-result. The latter, of course, really follows even the most malunited fractures. Böhler's former first assistant, Schnek, has modified this splint, so that it is neater, fits under the axilla better, and can also be used as an abduction splint. Böhler states: "I now challenge Magnus to produce or show X ray pictures of any cases which he has treated for three weeks by traction in bed with osseous union in good alignment." This challenge was thrown to Magnus because he stated in an earlier number of the same journal (Number 2, 1936) that Böhler's splint was worse than the disease: "I show you a Böhler's splint in illustrations and in the original. The patient came direct from Vienna and had been splinted for nearly three weeks. The fractured bones were displaced by a thumb's breadth. The condition of the man had not improved any more than would have been the case had he been spared the use of the splint." Against this, Böhler wrote that he could not be held responsible for wrong application of the splint, but was prepared to test his end-result statistics against those of Magnus, and he duly proceeded to do so. Speaking of spinal column fractures, Magnus has also criticized Böhler's work. "The treatment is expensive and time-robbing, and it is a nuisance for the patient. The treatment according to Böhler of the ordinary fractured spines with moderate displacement I hold principally as a mistake and I could not recommend it." Magnus treats such injuries with rest in bed, massage and so on, but no splinting. Böhler then proceeded to demonstrate a large number of illustrations to justify his own practice, and by showing photographs of cases treated by Magnus's method he put a very strong case against the latter (see Figure I). Although this is not stated by Böhler, it is interesting to note that Magnus, a professor of surgery, has so far had no good results with nailing fractures of the femoral neck, considers very few cases suitable for the Whitman method, and relies on traction in bed, for which method he claims good results! (*"Die Behandlung der Schenkelhals Fraktur", Deutsche medizinische Wochenschrift*, Number 24, 1936.)

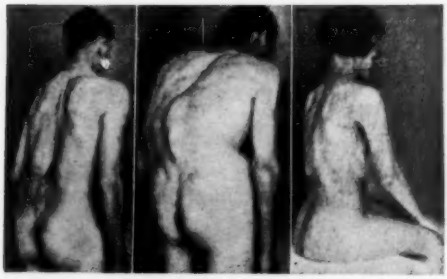


FIGURE I.

Magnus stated that all cases treated in a corset required as long a period of treatment as those treated by rest in bed and massage, that is, two to eight months in the corset. Böhler has seen severe cases which have been treated according to Magnus's principles and then later, after even weeks and months, have developed a kyphosis. Figure I, depicting a young girl who was treated by six weeks' recumbency and then massage, shows such a case, and it is not an isolated one. Magnus keeps patients lying on their back for four weeks; they are then allowed to walk after a further fourteen days and go to the massage department, where exercises are commenced. Böhler's patients get up on the second day after application of the corset, and usually leave the hospital at the end of the first week. The treatment therefore is not expensive or tedious, nor is it uncomfortable for the patient, who collects his own insurance and even works in the garden. In one case Böhler was called to a mountain-top, where a girl skier had a fractured spine (compression of the lumbar region). He reduced it simply up there, and although she had some initial paralysis, she was walking in a week or so. In short, he says: "Who could take the responsibility of risking permanent crippledom to a young person when the simple method of reduction and application of a corset is available?" Most surgeons would agree with Böhler. On the other hand, slight injuries can be supported by a light metal spinal brace, which is more comfortable. Another simple and very good method is to treat the fractured spine (especially in shocked patients) with recumbency, on a short "Dunlopillo" mattress, hyperextension being maintained by a "Dunlopillo" cushion or overhead sling (padded with one-inch "Dunlopillo") for three months or so. One can safely encourage any exercises except those which cause flexion of the spine. Perfect results can thereby be obtained. This

is very different from Magnus's treatment, which is really Lucas Championnière's applied to spinal column fractures, and therefore to be condemned unless used only in simple cases. In hospital practice throughout the world the treatment by early reduction (unless there is shock), immobilization in a plaster cast and early ambulation gives good results at a low cost.

THOMAS KING.

MODERN ASPECTS OF CORNEAL TRANSPLANTATION.

Ramon Castroviejo: "Keratoplasty", *American Journal of Ophthalmology*, Volume xvii, Number 10, October, 1934, page 932.

R. Castroviejo Briones: "*Queratoplastia: Estudio Experimental en Conejos con Corneas Leucomatosas*", *XIV Concilium Ophthalmologicum Espania*, 1934, T. 1–3, page 78.

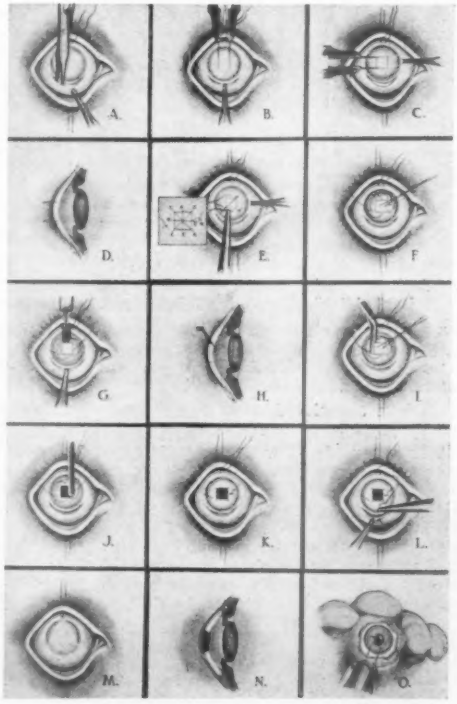
Ramon Castroviejo: "Keratoplasty: Report of Cases, with Special Reference to Complicated Ones", *The Journal of the Medical Society of New Jersey*, 1935.

R. Castroviejo Briones: "*Queratoplastia: Estudio Clinico de Catorce Casos. La Cornea de Feto como Material de Transplante*", *The Spanish-American Archives of Ophthalmology*, August, 1935.

Ramon Castroviejo: "Modern Aspects of Corneal Transplantation", from the Institute of Ophthalmology of the Columbia Presbyterian Hospital, New York.

Ramon Castroviejo: "Modern Aspects of Corneal Transplantation", reprinted from the Graduate Lectures before the American Academy of Ophthalmology and Otolaryngology, New York, 1936.

THE writer, having recently had an opportunity of viewing the remarkable corneal transplants of Castroviejo in New York and Tudor Thomas in London, feels that a short abstract of the six articles mentioned above in chronological order is of special interest, inasmuch as they reveal the surgical progress of one particular operation in the hands of the same surgeon over a period of three years. In these articles are given details of methods of diagnosis, including the transillumination method of the author and Mendosa, which have proved of great assistance in determining the site of the pupil and whether posterior or anterior synechia exists. Details of the preparation of the patient for operation and lists of special instruments required are also given. Various changes in operative technique are exhibited throughout the six articles, and the methods of after-care are also elaborated.



Unlike Tudor Thomas, Castroviejo favours a square instead of a round graft, and prepares it with a double-bladed knife, producing the necessary shelving edges with a keratome (see figure). He uses as material for the graft the eyes of still-born children in preference to the enucleated eyes of adults. It appears to the writer, having regard to the greater repair that occurs in an infant's cornea after inflammation as compared with that in an adult's, that it is logical to suppose that the younger the

donor, the more likely is the graft to maintain or regain its transparency. Castroviejo has also most clearly shown in human transplants that the corneas of donors with increased intraocular tension are definitely unsuitable for transplantation, but that the corneas from eyes containing a sarcoma are probably the most satisfactory type of adult material. He stresses over and over again the absolutely imperative necessity of avoiding an anterior synechia after the operation. He advises wide dilatation of the pupil with 3% atropine, commencing the day previous to operation, and, although in his last article he still advocates the use of conjunctival flaps to retain the graft in position, the writer found, when he visited New York this year, that the conjunctival flap method had been superseded by corneal stitching after the manner of Tudor Thomas. The reason why the conjunctival flap has been abandoned by Castroviejo is that the flaps occasionally adhere to the graft, causing one of two complications, either vascularization of the graft, which always ends in corneal opacity, or adherence to the graft, causing its dislocation when the flap is released.

He has made many alterations in the post-operative treatment until, finally, he believes that to leave the eyes untouched for six days after grafting is probably the most satisfactory procedure, and that provided that the pupil has a wide dilatation at operation, the risk of anterior synechia without further mydriasis is not very apparent.

Castroviejo finds that asepsis is absolutely vital, and that conjunctival cultures must be taken from both the donor and the recipient beforehand; but he has not found that blood grouping tests have revealed any practical information whatsoever.

One fact may be mentioned to show the ingenuity and impartiality of this author. Although he has been consistently successful with his double-bladed knife in commencing the cutting of the graft, yet when the writer was in New York he was experimenting on animals with a new suction trephine, which he thought might remove the disks in the donor and recipient with less trauma than by prevailing methods—a point stressed by Stallard in 1935. Possibly nothing will appear in the literature for some little time yet about this new feature, but it does show that new methods are being evolved and that refinements in technique will continue to appear. There is, however, no doubt that recent advances in technique have, in experienced hands, lifted keratoplasty out of the experimental stage into one of practical possibility, and that with wise choice of donor and recipient the prospects of a successful graft are very bright. As a result, there seems great hope of restoring useful vision to many inmates of blind asylums and similar institutions throughout the world. If each decade of this century produces a Gonin, Thomas or Castroviejo, then I feel certain that the cherished hopes of the International Association for the Prevention of Blindness will be realized before the next century appears.

J. BRUCE HAMILTON.

DANGERS OF ENDOSCOPIC PROSTATIC RESECTION.

H. Viethen: *"Die Gefahren der endourethralen Elektrotomie, und ihre Verhütung"*, *Zeitschrift für Urologie*, Volume xxxi, Number 5, May, 1937, page 319.

THE author considers that, since transurethral resection has been used in Germany for five years, the question of its dangers should now be fully reviewed. Instruments which do not allow the actual cuts to be made under visual observation through an optical system are condemned.

Risks associated with perurethral resection may be general or local.

1. *General Risks.*—(*a*) Cardiac complications. As far back as 1925 it was estimated that 42% of patients with prostatic hypertrophy had some form of cardiac disease. Such figures make us pause, in view of the large numbers of people who are nowadays clamouring for treatment by perurethral resection. The danger is that of extra load on the heart as a result of infection and toxic absorption from coagulated albumin. The latter factor is of marked importance following extensive resections. It is at its greatest height on the second and third days after operation, as shown by increased pulse rate and other signs of cardiac failure. In all suspected cases digitalis therapy should be employed for a week or two before operation. With advanced myocardial degeneration, the resection should always be carried out in two or three sessions.

(*b*) Post-operative ascending infection. Although the common chronic urinary tract infection is not a contraindication to resection, frank purulent infection, especially if accompanied by pyrexia, demands preliminary catheter drainage, bladder irrigations and antiseptics by mouth.

2. *Local Risks.*—(*a*) Hæmorrhage. The proper current to choose is one which affords efficient cutting with a moderate intensity of coagulation. Should the cutting loop be moved through the prostatic tissue too quickly, a tearing effect is the result, with consequent troublesome bleeding. Secondary hæmorrhage occurring ten to fourteen days after operation seldom gives much trouble, and is easily controlled by the use of an inlying catheter and hot irrigations.

(*b*) Perforation of the bladder wall. Insufficient filling of the bladder may result in the thin posterior wall being injured by the loop, since under such conditions the posterior wall has not been adequately separated from the bladder neck region. Perforation into the peritoneal cavity is the result, and calls for immediate abdominal section.

(*c*) Perforation of the urethra. Perforation of the urethra may occur in the anterior part of the *pars prostatica* or in the *pars membranacea*. Such injuries may be followed by wide and spreading phlegmonous inflammation. The *colliculus seminalis* is the most distal limit during the removal of strips of prostatic tissue, and the cuts should not actually reach this structure.

R. J. SILVERTON.

ELECTRO-RESECTION FOR BLADDER NECK OBSTRUCTION.

Kurt Tzschirntsch: "*Zur Elektroresektion bei obstruirenden Blasenhalsveränderungen*", *Zeitschrift für Urologie*, Volume xxxi, Number 5, May, 1937, page 305.

THE interesting questions for urologists are: What are the results of this new method, and what is the proportion of failures? The author's experience is in 44 cases extending over a period of two and a half years. Spinal anæsthesia is the best method. A German-made loop instrument is used. Electrical and other technical defects interfered very little with the course of the operations. Blockage with clots during operation gave trouble only seldom. A false passage was made in one case, so immediate cystotomy was carried out. A few months later, transurethral resection was successfully performed. Visibility is certainly interfered with at times by bleeding, but this was never severe enough to demand cystotomy for its control. In 22 of the 44 cases there was complete retention of urine prior to operation, while the remaining patients had varying amounts of residual urine. In 23 cases there was a concentric adenomatous hypertrophy of the collar type, and in 10 cases the lateral lobes were grossly enlarged. In 28 cases one resection sufficed for cure, while in 13 a second resection was necessary. Only in two cases was a third resection required. Of the patients whose ages ranged from sixty to seventy-two years, that is, in 33 of the series, only one died, and this was an exceptional case, the patient having had previous unskilful treatment followed by the development of a suprapubic fistula, and was a morphine addict. It is notable that among the remaining 11 patients, whose ages ranged from seventy-three to eighty-five years, there were seven deaths.

R. J. SILVERTON.

FAILURES AND IMPERFECT RESULTS IN TRANSURETHRAL PROSTATIC RESECTION.

E. Kraas: "*Fehler und Misserfolge bei der endourethralen Prostataresektion*", *Zeitschrift für Urologie*, Volume xxxi, Number 5, May, 1937, page 311.

KRAAS considers that inordinately great hypertrophy of the prostate must be looked upon as a contraindication to endoscopic resection, since during the operation uncontrollable hæmorrhage is likely to be encountered, with the consequent necessity for immediate suprapubic cystotomy. Furthermore, there is a tendency in such cases to secondary hæmorrhage. Where there is much enlargement of the lateral lobes intravesically, the technical difficulty of removing a sufficient quantity of adenomatous tissue is very great, and here again prostatectomy is the operation of choice. A roomy canal must be made through the adenomatous tissue, and more superficial resection is useless. In the cysto-urethroscopic image one should be able to see, after resection, from the *colliculus seminalis* right back to the *trigonum vesicæ*, and projections of the lateral lobes impinging on this open tunnel must be cleared away. Infection should be removed before operation. During the resection as little coagulation as possible should be carried out, since this itself leads to infection. Nevertheless, on account of its better hæmostatic power, a spark-gap instrument is to be preferred to a tube machine.

R. J. SILVERTON.

THE IMPORTANCE OF CHOLANGIOGRAPHY IN PRACTICE AND RESEARCH.

Dr. Fr. Bernhard (Chirurgischen Universitätsklinik, Giessen): *"Die Bedeutung der Cholangiographie für Praxis und Forschung"*, *Der Chirurg*, March 15, 1937.

THE opportunity of obtaining a radiographic picture of the bile duct tree which is offered by a drainage tube in the common duct after choledochotomy, is one that should be more generally made use of in the interests of patients. It is entirely harmless, and Bernhard has made it a routine measure to obtain such radiograms in all cases of choledochotomy performed in the last two years.

Fifteen to twenty cubic centimetres of "Thorotrast" are injected into the drainage tube by syringe. When much dilatation of the duct tree is present, more can be used; but in any case the injection is stopped at once on the occurrence of any feeling of pressure. The injection of morphine beforehand is not wise, as it is apt to cause spasm of the sphincter and a possibly faulty picture. "Thorotrast" has not the disadvantages

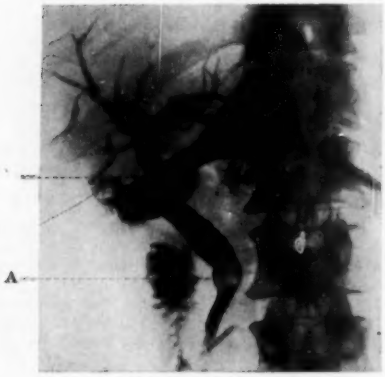


FIGURE I. A = overlooked stone in common duct.

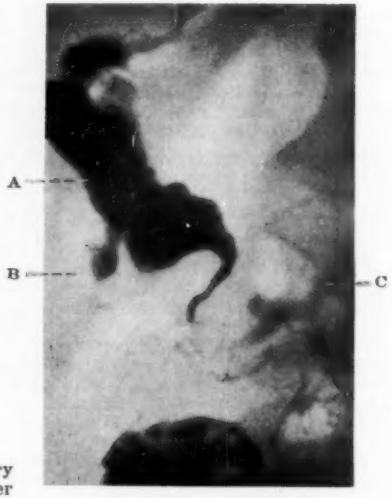


FIGURE II. A = common duct; B = anastomosis between common duct and duodenum; C = duodenal cap filled with air.

in the bile system that it has in the urinary passages. It does not penetrate into the liver tissue, and does not become precipitated in flakes as it is inclined to do in the acid urine medium. When cholecystitis is acute and the dilatation of the ducts great, it is perhaps wiser to use "Uroselectan-B" or "Abrodil", in case flocculation should occur. Should it be retained in the ducts, as shown by radiography, for more than twenty-four hours, it should be washed out with normal saline solution.

Although this method can be used with safety as a routine, it is especially helpful in those cases in which a free bile flow persists beyond the second week, and in those cases in which the clamping of the drainage tube produces a feeling of pain or pressure within the bile system. Cholangiography will nearly always reveal the cause of the trouble in these circumstances and thus enable it to be properly treated. If, for instance, it is demonstrated that the contrast medium passes quickly and easily into the duodenum, the surgeon is freed from the usual doubts about removing the tube, and may remove it with an easy mind. Its greatest value lies in its demonstration of pathological conditions overlooked or not found at the operation. Figure I shows a common duct stone which was missed at operation. These stones are not radio-opaque and need a contrast medium to demonstrate them. It is necessary to guard against mistaking an air bubble in the medium for a stone, and in case of doubt a repetition of the radiogram will make the condition definite.

Figure II shows a narrowing of the duct owing to pressure by the enlarged head of the pancreas, after a second operation of choledocho-duodenostomy had been performed

to relieve the obstruction. It is in such cases that the decision to operate can be made early before the stricture has caused any grave liver damage. Obstructions of the lower common duct are more likely to be due to inflammatory swelling, or to spasm of the sphincter region, than to pancreatitis. It is interesting in this respect to reflect that in some 10% of films of such cases the contrast medium is found filling the pancreatic duct on cholangiography. It will be found in some films of the one patient, but not in others of the same patient. This is of interest as suggesting that regurgitation from the common duct into the pancreatic duct does not carry the danger of acute pancreatitis which has been claimed for it.

ARTHUR E. BROWN.

PRIMARY BLADDER CLOSURE IN SUPRAPUBIC PROSTATECTOMY.

T. Wohlleben: *"Primäre Blasennaht bei suprapubischer Prostatektomie", Zeitschrift für Urologie,* Volume xxxi, April, 1937, page 270

THE majority of surgeons after operations on the urinary bladder use suprapubic drains, sometimes large, while after prostatectomy tampons in the prostatic cavity are employed as well. The rationale of such drainage is that fairly large clots can be easily evacuated, and that the risk of infection of the upper urinary tract is diminished. In spite of such time-honoured considerations, there is another school of surgeons at present who think that the bladder should be sutured primarily after most bladder and prostatic operations. Wohlleben states that it is indeed surprising, considering the excellent reports from the group of surgeons referred to, that the method of primary closure has not gained more adherents. Even in those cases where the prostatic cavity is carefully packed, the risk of bleeding remains. In Wohlleben's experience, hæmorrhage after prostatectomy where packing is not carried out is not alarming, and can be dealt with through an indwelling urethral catheter, while if bleeding later becomes severe, provided that the proper instruments are used, clots can be evacuated by suction *per urethram* and the hæmorrhage controlled by hot irrigations. The author does not attempt to discuss the continued efforts made by others to control bleeding by direct ligation of vessels in the prostatic bed, but contents himself with reporting that he has found very copious hot saline irrigations of sufficient value for this purpose. After several litres of such hæmostatic irrigations, a 26 Charrière de Pezzer drain is passed with a sound as guide out from the bladder through the urethra, so that the mushroom end lies snugly in the depth of the prostatic bed. The central portion of the mushroom end is cut away so that subsequent aspiration of clots, should this become necessary, is facilitated.

The bladder is sewn up firmly in two layers. A small drain is left in the prevesical space for five days, and the de Pezzer drain is left in the urethra for about one week. After four or five days the urine usually appears free from blood. At this time the de Pezzer drain is spigotted, and uncorked as soon as the slightest desire to urinate occurs. When the drain is to be removed, a little intravenous "Evipan Sodium" is given. It is found that the drain slides out fairly easily.

The patients are allowed up much sooner than is usually considered advisable. Two days after the operation the patient is allowed up for two periods of half an hour each, and gradually for longer periods on the following days. The patient is, as a rule,, allowed to get about a little on his feet on about the fourth day.

R. J. SILVERTON.

Reviews.

GENITAL ABNORMALITIES.

Genital Abnormalities, Hermaphroditism and Related Adrenal Diseases. By H. H. YOUNG, M.A., M.D., Sc.D., F.R.C.S.I., D.S.M.; 1937. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 690, with 534 illustrations. Price: 45s. net.

THIS book is an extremely lucid and well-written work on a very difficult and obscure subject. The author opens with a chapter on the origin of the term hermaphrodite, and

traces the references to the condition in the classics and the literature of the Middle Ages, with reproductions of the better known statutes and paintings that have been preserved from ancient times.

The embryology of hermaphroditism is discussed in such a manner as to render the development of genital abnormalities easy to understand. The various types of hermaphroditism are described and discussed in detail in a most convincing manner, each chapter being illustrated by cases drawn from the author's own experience. The very fact that he is able to quote eighteen personal cases serves to demonstrate the extent of this experience. The cases consist of eight male pseudo-hermaphrodites, four female pseudo-hermaphrodites and five cases where the true sex was not determined. One case of true hermaphroditism is presented in detail and the diagnosis proven microscopically, and the nine other cases recorded in the literature are reviewed. One of these was so perfectly developed that the author states that it would not be extravagant to claim that auto-fertilization might have been brought about, had not the testis been removed in the belief that it was undeveloped.

Vaginal abnormalities in hermaphroditism are classified, and mention is made of the fact that it is possible to investigate many of these with the irrigating cysto-urethroscope, especially in those cases where the vagina opens into the urethra. In this event the instrument can be introduced into the vagina and the presence of a *cervix uteri* demonstrated. Various plastic procedures for the reconstruction of the vagina are described.

The adreno-genital syndrome is treated very fully, both hyperplasia of the adrenal cortex and tumours of the cortex being discussed. The best technique for the exposure and removal of the adrenal is discussed, and this section is illustrated with a series of beautiful plates showing each stage of the operation.

Hyper- and hypo-genitalism have each a chapter devoted to them, and also virilism, each condition being exemplified by case records. The presence of prostates in the female and gynæcomastia are dealt with, as well as the more common conditions, such as hypospadias, epispadias and extroversion of the bladder. Abnormalities of the epididymes, *vasa deferentia* and seminal vesicles receive their quota of attention, and *atresia ani urethralis* brings to a close this impressive array of genital abnormalities.

The final chapter is devoted to a discussion of the relation of the genital tract to the endocrine glands, and to endocrine therapy. This chapter is well worth studying, especially in view of the somewhat haphazard manner in which endocrine therapy is on occasion employed.

To summarize, this book is an exhaustive work on all those conditions mentioned in the title, written by a man who is obviously a master of his subject. It will be of interest especially to the urologist and the gynæcologist, to whom it will be very useful as a book of reference, more particularly as all the surgical procedures employed are described in detail, together with the results of these methods.

THE LABORATORY DIAGNOSIS OF SYPHILIS.

The Theory, Technic and Clinical Interpretation of the Wassermann and Flocculation Tests with Serum and Spinal Fluid. By H. EAGLE, M.D.; 1937. St. Louis: The C. V. Mosby Company. Australia: W. Ramsay (Surgical) Proprietary Limited. Royal 8vo, pp. 440, with 49 tables. Price: 30s. net.

THE importance of the laboratory tests for syphilis increases rather than decreases with the years. An intelligent interpretation of laboratory reports by the physician or surgeon is impossible unless he be familiar to some degree with the numerous sources of error in the technique, and aware of the significance of the terms used in the reports. Further, despite the experience of forty years since the Wassermann test was instituted, there are still problems, of which outstanding ones are: What constitutes the optimum Wassermann technique; which is the optimum flocculation test; and which of these two is the method of choice? These questions are dealt with in this book with a wealth of detail, lucidly, intelligibly, and from the background of years of experience in a large Wassermann laboratory intimately associated with a large syphilis clinic.

Two groups of workers are deeply concerned with the progress of the serology of syphilis: the laboratory workers responsible for the tests, and concerned with their performance, rationale and scientific basis; and a larger group, almost the whole medical profession, the members of which must utilize in practice the results furnished by the

laboratory. Between these groups there is too often a gap of misinterpretation. This book, for any who will read it, should do much to bridge this gap.

Serologists themselves are sometimes in doubt as between endeavours to standardize the Wassermann test in every laboratory, and inclination to discard it in favour of the newest flocculation reaction. A study of these pages will resolve much of the prevailing confusion. The author makes it quite clear that, although there is a multiplicity of methods, fundamentally all the Wassermann tests are one; and although the many flocculation tests have many names, essentially they are one, and are all dependent on the laws governing the reactions of colloids, whereby aggregation, or precipitation, or flocculation—whichever term is most appropriate—is brought about. The value of any one of these tests must ultimately depend on two criteria: (1) its specificity for syphilis, that is, the absence of positive reactions in the presence of any other conditions; and (2) its sensitivity, that is, the percentage of syphilitic infections detected. Dr. Eagle discusses the source and nature of the various reagents necessary for each test, and the difficulties as well as the virtues which belong to each method. There is a note of directness, even dogmatism, in his writing, and some impatience with old methods (for example, any Wassermann technique without ice-box incubation). At the same time, the case for the newer methods is well made out, and British serologists who have adopted a rather conservative attitude as compared with workers in the United States of America, will feel constrained to meet the challenge by trying out in parallel the old and the new.

The author makes quite clear some points which should be realized by every medical practitioner; for example, the greatly enhanced value of the double test (complement fixation and flocculation) over either test alone; and, again, the high degree of sensitivity of Wassermann tests of the cerebro-spinal fluid and their consequent paramount importance for prognosis and treatment where, as always, there is a possibility of neurosyphilis.

The book answers just those questions which are constantly being put to the laboratory worker by the practitioner. It shows the great advances that have been made even since the last conference with practical tests arranged by the League of Nations (1930), and it asks for the setting up of a permanent organization to arrange trials of methods and to coordinate tests in the future.

The book is well produced, is commendably free from misprints and inaccuracies (although one incorrect symbol, *minus* instead of *plus*, occurs in Table XXXIX under the heading "Relative Agreement"), and can be recommended to all those who desire to understand this subject and keep abreast of current practice.

A SHORT TEXT-BOOK OF SURGERY.

A Short Textbook of Surgery. By C. F. W. ILLINGWORTH, M.D., F.R.C.S.; 1938. London: J. and A. Churchill Limited. Medium 8vo, pp. 710, with 8 plates and 179 text-figures. Price: 21s. net.

THE author writes a very modest preface to this extremely readable text-book. It is eminently suitable for students; but we think it is a little brief for those studying for most post-graduate degrees. Each section commences with an account of the routine examination of the part concerned, after which the pathology of the region is dealt with concisely, and usually with a good illustration of the condition. Treatment is only indicated in broad outline and principle. No operative details are given—an excellent idea in a text-book of this nature, which must be kept down to a convenient size.

The book is most up to date in treatment, and the author gives the orthodox views which would be acceptable to any examiner. We can thoroughly recommend it as a standard text-book.

THE ART OF SURGERY.

The Art of Surgery: A Text-Book for Students and Practitioners. By H. S. SOUTTAR, D.M., M.Ch., F.R.C.S.; Third Edition; 1937. London: William Heinemann (Medical Books) Limited. Imperial 8vo, pp. 640, with illustrations. Price: 30s. net.

IN this new edition Souttar's "Art of Surgery" has not been materially altered from previous editions. The title does not betray any definite claims for the work, which

in effect can only be regarded as a text-book suitable for a final year medical student or a guide for coaching. For this purpose it is attractively and helpfully written. The text is well arranged, and the matter synoptic, often, however, to the point of being inadequate. It is characteristic of the incomplete way in which many subjects are handled that antiserum is not even mentioned in the paragraph on gas gangrene.

Every page has marginal sketches, which are illustrative of the main points under discussion. There is a lot of originality and ingenuity in this aspect of the book. There are some beautiful colour plates, the inclusion of which, as in so many such works, is justified rather by their artistry than their usefulness.

The practising surgeon will not find "The Art of Surgery" one of those repositories of information, of discussions of common difficulties, or of references to contemporary literature, for which he may seek. Teachers, however, may well be struck by its many good features to commend it for student use.

ORTHOPÆDIC SURGERY.

A Practice of Orthopædic Surgery. By T. P. McMURRAY, M.B., M.Ch., F.R.C.S.; 1937. London: Edward Arnold and Company. Demy 8vo, pp. 479, with illustrations. Price: 21s. net.

THIS book is dedicated "To the memory of Sir Robert Jones, the father of orthopædic surgery", and, as one would expect in a book from the Liverpool School, breathes the spirit of his genius.

As stated in the preface, the author is concerned mainly with basic principles, operative details being only briefly included in the text. The subject of fractures has been excluded, as it is considered, quite correctly, that this requires a separate volume. The book can be highly recommended to the student and the general surgeon, but possibly the orthopædic specialist would require more details of newer methods of surgical treatment and more discussion of debatable subjects. Everything recommended in the book has been thoroughly tested and found effective, and nothing experimental or doubtful is included. The book is generously illustrated with plates and skiagrams, and is well printed and of a convenient size.

THE COLLAPSE THERAPY OF PULMONARY TUBERCULOSIS.

The Collapse Therapy of Pulmonary Tuberculosis. By J. ALEXANDER, B.S., M.A., M.D., F.A.C.S.; 1937. London: Baillière, Tindall and Cox. Baltimore: C. C. Thomas. Demy 4to, pp. 720, with 367 illustrations. Price: 67s. 6d. net.

IN 1926, John Alexander wrote a small monograph, "The Surgery of Pulmonary Tuberculosis", which contained an account of the surgical practice of that period, preceded by a brief historical review. Since 1926, progress in the surgical treatment of pulmonary tuberculosis has been so rapid that little beyond the historical section of that monograph has survived the interval of twelve years. The rest has been rewritten and elaborated in collaboration with several of his colleagues, under the title "Collapse Therapy of Pulmonary Tuberculosis".

The book is based upon a study of 1,800 cases, and combines thoroughness, enterprise and personal experience with a critical review of numerous articles and books, as well as observations of the work of other men and other countries. Such a notable contribution to thoracic surgery should be a pride not only to the author, but to the publisher, who has blessed the work with excellent format, letterpress and illustrations.

In the first chapter Alexander summarizes the admirable papers of S. L. Leslie and R. S. Anderson (*American Journal of the Medical Sciences*, 1937), showing the gratifying results of surgical treatment in 78·8% of 1,124 patients, in 52·8% of whom the disease was far advanced. Such results studied impartially must convince the most sceptical physician of the rôle of surgery in the modern treatment of pulmonary tuberculosis. Alexander rightly stresses the importance of special training for thoracic as for neurological surgery. The general surgeon who attempts much of either without adequate and intensive training is doomed in the long run to do more harm than

good. In the interesting chapter on the evolution of surgical therapy, which appeared in the 1926 monograph, honour is given to the pioneers of all countries.

The lucid chapter on the physiological principles and the pathology of collapse therapy should be of special interest to two main groups: first, those who "do not hold with surgery for pulmonary tuberculosis", and, second, those who would operate indiscriminately with a deplorable lack of judgement and knowledge. To decide whether operation is indicated, and, if so, what type of operation is needed, the surgeon must be trained to examine and assess the patient himself and to recognize contra-indications, as well as indications, for operation; it betokens negligence and ignorance to depend solely upon radiograms, an abstract of the history and the physician's views. The physician has a right to receive a considered and intelligent opinion from the surgeon in this as in other branches of surgery. If Alexander's advice on these points is taken to heart and practised, there will be less "carpentry" and more real surgery in this type of work, with a corresponding decrease of bad results which bring disrepute upon the operative treatment.

Many readers will be surprised to find, in the discussion on the choice of operation, that Alexander prefers phrenic paralysis to pneumothorax in certain cases for which physicians with small experience of the phrenic nerve operation would advise pneumothorax induction; for example, he finds that phrenic paralysis is generally the best operation for hæmoptysis which resists non-surgical treatment. The occasional thoracic surgeon who usually decides on permanent phrenic paralysis by avulsion of the nerve would do well to ponder Alexander's words in favour of temporary paralysis by crushing. In this preference Alexander is in accord with most thoracic surgeons of experience.

The various operations and combinations of operations, with indications, contra-indications and management, are fully and thoroughly discussed. Illustrative cases show that, with careful selection and control, bilateral collapse therapy for bilateral disease will snatch many a brand from the burning.

There is a useful chapter on anæsthesia. Chloroform is rather summarily condemned; but such an experienced thoracic surgeon as Morriston Davies still prefers it, supplemented by nerve block with "Novocain", to the newer anæsthetics; the reviewer has personally watched Davies at work, has seen the patients afterwards, and is convinced of its virtues. "Cyclopropane" receives a bare notice, and no mention can be found of intravenous "Evipan" or "Pentothal Sodium" as a preliminary to gas and oxygen anæsthesia. Some of us with only a small experience can speak well of this combination.

The two concluding chapters deal, with the author's usual thoroughness, with tuberculous empyema and tuberculosis of the thoracic wall respectively.

This book is already amongst the classics. Physicians and surgeons who are interested in the treatment of pulmonary tuberculosis must read and digest it.

OBSTETRICS.

Obstetrics. By G. FITZGIBBON, M.D., M.Ch., B.A.O., F.R.C.P.I., F.C.O.G.; 1937. Dublin: Browne and Nolan Limited. Demy 8vo, pp. 495, with 167 illustrations. Price: 16s. net.

ANY book emanating from the famous Rotunda School must command the interest of those practising obstetrics. Gibbon Fitzgibbon, already noted for his advocacy of the trial labour, has summarized his extensive clinical experience in a small text-book of obstetrics which clearly defines both the conditions constituting normal delivery and those complications which require careful supervision.

There is a concise description of the fundamentals of anatomy and physiology in their relationship to pregnancy and labour. One is surprised, however, to note that the author still favours the use of the binder during the puerperium, and also advocates the patient rising at an earlier period than is the custom here. He places little emphasis on the possible complications associated with occipito-posterior presentations and describes the conservative methods used in such cases. The chapter on defective uterine action is very well presented and gives a clear clinical classification of this troublesome condition. In the treatment of pelvic disproportion the author has apparently changed his views, and now appears to favour induction rather than a trial of labour at term for many border-line cases. The reasons put forward in support of this change are not over-convincing, but the description of the clinical management of such cases is

exceptionally well done. The chapter on the care and management of the infant might have been expanded in many directions.

In its unpretentious format this book may not appear attractive to those who have been more accustomed to the lavishly illustrated American treatises, but throughout the text one can appreciate the sound clinical judgement of an ex-master of the Rotunda Hospital. It can be thoroughly recommended as a practical guide, both for students and for those in general practice.

AN INTRODUCTION TO OPHTHALMOLOGY.

Introduction to Ophthalmology. By P. C. KRONFIELD, M.D.; 1938. Baltimore: C. C. Thomas. Medium 8vo, pp. 340, with illustrations. Price: $3.50 net.

PROFESSOR KRONFIELD, of the Peiping Union Medical College, has written a book which "endeavours to formulate the principles underlying that portion of ophthalmology which is a necessary part of basic medical education".

There is a brief anatomical description of the eye and its adnexa, followed by the usual classification of diseases. In the text the author only describes the commoner conditions as examples of general pathological processes, but he gives a brief description of many other diseases in the index. At the end of each chapter there are a few references for those who wish for more detailed information.

The book is written in a manner that shows the application to the eye of general physiological and pathological principles. Where there are no special features to be described (as, for example, in tumours of the lids) the subject is omitted altogether. The book is thus not a treatise on ophthalmology, but is truly an introduction, and as such should be very useful to students who wish for some general information on diseases of the eye. It is written in a simple conversational style, and it should be easy for the student to acquire a great deal of information by once reading it. The general arguments are not held up by laborious classifications. Irritating descriptions of rare diseases are omitted.

The book can be confidently recommended for undergraduates. It should be read while they are receiving practical instruction in eye diseases.

Proceedings of the Royal Australasian College of Surgeons.

VICTORIAN HALF-DAY MEETING.

THE Victorian State Committee arranged a half-day meeting at the Royal Melbourne Hospital on Wednesday, November 9, 1938. The following programme was presented:

Theatre on the First Floor.

Operations—

2 p.m. to 3.15 p.m.—R. W. Chambers: Myomectomy.

3.15 p.m. to 4.30 p.m.—G. C. Scantlebury: Ferris Smith operation.

Demonstration in ante-room: John Shaw and T. G. Millar.

Theatre on the Second Floor.

Operations—

2 p.m. to 3.15 p.m.—E. E. Price: Osteotomy of radius for non-congenital synostosis of forearm.

3.15 p.m. to 4.30 p.m.—Henry Searby: Operation for inguinal hernia.

Demonstration in ante-room—E. E. Price: Some principles in the treatment of compound fractures of the lower end of the humerus.

Theatre on the Third Floor.

Operations—

2 p.m. to 3.15 p.m.—J. T. Tait: Operation for renal calculus.

3.15 p.m. to 4.30 p.m.—W. A. Hailes: Operation for the treatment of a proliferating tumour of the bladder.

NEW SOUTH WALES ANNUAL DINNER.

A DINNER arranged by the New South Wales State Committee was held at the Royal Automobile Club, Sydney, on December 7, 1938.

Professor Eric Ashby, D.Sc. (London), D.I.C., delivered an address entitled "Darwin and Health".

GORDON CRAIG MEMORIAL LIBRARY.

THE attention of Fellows is drawn to the following papers which have recently been fully translated from foreign literature:

"*Pancreatitis Acuta*", *Acta Chirurgica Scandinavica*, Volume lxxv, 1934, pages 373 to 415.

"Extending the Combat of Portio-Carcinoma with the Aid of the Colposcope" (Munich Medical Association), *Münchener medizinische Wochenschrift*, Number 47, 1937, page 1885.

"Grafts in Caoutchouc (Nouva Carne) Observed after 25 Years", by Professor D. Fieschi, *Revue de Chirurgie*, January, 1938, pages 1 to 36.

"The Colposcopic Picture of Carcinomatous Coatings", by Ernst Preissecker, *Wiener klinische Wochenschrift*, 1929, Number 42.

"New Therapy for the Partially Deaf", by Professor Emil Glas, *Wiener medizinische Wochenschrift*, Number 6, 1938.

"On the Thyreotoxic Crises and Their Treatment, with Especial Regard to Iodine Treatment", by E. J. Wijnbladh, *Acta Chirurgica Scandinavica*, Volume lxxix, 1937, pages 507 to 544.

"On the Treatment of *Keratoconjunctivitis Sicca*", by Dr. W. Meisner, *Zeitschrift für Augenheilkunde*, Volume xciv, February, 1938.

"Contribution to the Chapter of Inflammatory Pseudo-Tumours of the Orbita, à propos of Two Cases of Unilateral Exophthalmos", by Hans Souttar, *Klinische Monatsblätter für Augenheilkunde*, Volume c, 1938, pages 29 to 42.

Copies of these translations are available to Fellows.

Once again the Council wishes to remind Fellows that the following services are available in the Gordon Craig Library:

1. Fellows may receive on loan any text-book, monograph, journal or reprint available at the College.
2. Fellows resident in the Commonwealth of Australia will not be permitted to retain a book for more than one month, and Fellows resident in the Dominion of New Zealand for more than two months.
3. Books not returned at the expiration of this time will be charged to Fellows.
4. No Fellow will be allowed to borrow more than two books or two journals, or one book and one journal, at one time without special permission.
5. Translations from foreign journals received in the Library will be supplied to Fellows free of charge.
6. Photostatic copies of articles and illustrations appearing in text-books, monographs, medical and surgical journals received at the College will be supplied to Fellows free of charge.
7. References will be supplied to Fellows of the College who are writing papers. Information concerning articles published on any particular subject, or by any particular author, will be supplied. Should the journal required not be obtainable in the College Library, facilities are available whereby Fellows may be informed whether the journal they require is available in Australia, and, if so, at what library.

Fellows are invited to avail themselves of these services.

H. G. WHEELER,
Secretary.

CONGRESS OF THE PAN-PACIFIC SURGICAL ASSOCIATION.

A CONGRESS of the Pan-Pacific Surgical Association will be held in Honolulu on September 15 to 28, 1939.

This will be the third meeting of the Pan-Pacific Surgical Association, the two former meetings having also been held during 1929 and 1936 in Honolulu. An invitation is extended to all surgeons to meet in Honolulu outstanding men from countries of the Pacific area, including Australia, New Zealand, China, Japan, Java, Canada and the United States of America, for an interchange of surgical thought and for the purpose of bringing about better understanding through personal contact among the surgeons of these countries.

There will be sections in fractures and orthopædics, general surgery, gynæcology, motion pictures, neurosurgery, ophthalmology, oto-laryngology, Röntgenology, plastic surgery, thoracic surgery and neurology, with outstanding men as chairmen for the United States of America and equally prominent men as chairmen for the Australasian section. The congress affords not only participation in interesting scientific papers, but a most enjoyable vacation in the "Paradise of the Pacific".

Communications for information should be directed to George W. Swift, M.D., 902 Boren Avenue, Seattle, past president of the association; Frederick L. Reichert, M.D., Standford University Hospital, San Francisco, programme chairman for the United States of America; Howard Updegraff, M.D., 6777 Hollywood Boulevard, Los Angeles, programme vice-chairman; Forrest J. Pinkerton, M.D., secretary-treasurer of the association, Young Building, Honolulu, Hawaii; or H. G. Wheeler, secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne.

Notices.

THE BRITISH POSTGRADUATE MEDICAL SCHOOL.

THE College wishes to draw attention to the announcement of the British Postgraduate Medical School on page xxxii of the advertisements.

NEW DEVELOPMENTS IN SURGICAL EQUIPMENT.

THE attention of Fellows is drawn to pages xx and xxviii among the advertisements, which illustrate some recent developments in surgical equipment. The Editorial Committee is responsible for the selection of the equipment illustrated thereon. The publishers will be pleased, whenever possible, to supply the names and addresses of the manufacturers to anyone requiring such information.

Editorial Notices.

EDITORIAL communications should be addressed to the Chairman of the Editorial Committee, 57 Collins Street, Melbourne, or to any member of the Editorial Committee. It is understood that original articles forwarded for publication are offered to THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY solely, unless the contrary be stated.

Reprints can be supplied at cost price; the minimum number is fifty copies. Orders for reprints must be given when the proof is returned.

Exchange journals should be addressed to the Honorary Librarian, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, Victoria, Australia.

Business communications and remittances should be addressed to Butterworth and Co. (Australia) Ltd., 8 O'Connell Street, Sydney.